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A STUDY OF THE FASCIAL SPACES OF THE FOOT AND THEIR BEARING ON INFECTIONS

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THE memorable work of Kanavel on the fascial spaces and tendon sheaths of the hand has given great impetus to a more thorough study of infections of the hand and with it better results from treatment properly applied. For some reason, no similar study has been made of the foot. Possibly this is due to the fact that foot infections are less common than hand infections because of the protection afforded by the wearing of shoes. Again it may be due to the fact that the clinical problem presented differs somewhat in the two cases. In that of the hand, there is, in addition to the immediate danger to life and the length of convalescence, an important economic problem concerned with the preservation of function. The hand is a very delicately balanced mechanism capable of carrying out the most intricate and skillful of movements—so important economically.

The foot is not so delicately adjusted nor is it of such direct importance in economic life. The foot is chiefly concerned with body support and locomotion, functions which, though they are important enough, are more easily preserved following infections than those of the hand. It makes little difference if the flexor tendon to one of the toes is functionless, but a similar condition in the hand would be of utmost importance. However, it is just as essential to get the patient with an infected foot well and back to work at the earliest possible moment as it is the one with an infected hand, so that from the standpoint of length of convalescence, as well as immediate danger to life, the foot becomes just as important as the hand.

This study was undertaken with the feeling that a better knowledge of the anatomy of the foot, particularly of the fascial spaces, might lead to a better understanding of foot infections, with lessened immediate mortality, shortened convalescence, and possibly better preservation of function as regards body support and movement. Two methods of study were used: first, careful dissection of the human foot to determine the extent and boundaries of the various fascial spaces; second, injection of the spaces with gelatin or paraffin to outline them and determine the route of spread from one to the other.

DISSECTIONS

Careful dissection of fresh and preserved human material reveals the presence of four potential median spaces on the plantar side of the foot, which may be conveniently designated as M_1, M_2, M_3, and M_4. In addition, a lateral space deep to the abductor digiti quinti, a medial space deep to the abductor hallucis, and spaces along the lumbrical muscles may be demonstrated. There are also two dorsal spaces—subcutaneous and subaponeurotic. Again, there is a space

between the superficial and deep calf muscles which is brought into relation with the foot by the long flexor tendons behind the medial malleolus, and a space deep to the fascial sheaths of the peronei, the tendons of which extend behind the lateral malleolus toward the foot. The extent and boundaries of these spaces may be seen in Figures 1 to 4, which are drawings of actual dissections, the first being a superficial view and each succeeding one at a deeper plane but drawn in the same proportions. The relations between these spaces can be further studied by referring to the drawings of the sagittal and cross sections (Figs. 5 to 7).

The removal of the skin and superficial fascia exposes the plantar aponeurosis in which a thick, middle portion over the flexor digitorum brevis, a thinner portion over the abductor digiti quinti (calcano-metatarsal band), and a very thin, indistinct portion over the abductor hallucis muscle can be distinguished. The middle portion consists of transverse and longitudinal fibers, the latter lying more superficially. Opposite the middle of the metatarsal bones, it divides into five slips extending to the bases of the toes. These slips are joined by ill defined transverse fibers or superficial transverse metatarsal ligaments which help to support the webs of the toes. Each slip splits into two divisions which are attached to either side of the metatarsophalangeal articulation and the base of the first phalanx. In addition, some of the longitudinal fibers extend into the skin of the toes and more particularly into the superficial layer of the digital fascia as described below.

MEDIAN PLANTAR SPACE M1

M1 lies between the central part of the aponeurosis superficially and the flexor digitorum brevis muscle deeply (Figs. 1, 5, 6, 7). The lateral and medial boundaries of this space are connective tissue downgrowths from the plantar aponeurosis which are continuous with the corresponding boundaries of M2 described below. This space is incompletely subdivided into four anteroposterior compartments by three very thin connective tissue septa which are reflections of the fascia on the deep side of the aponeurosis and the superficial side of the flexor digitorum brevis muscle. A compartment lies proximal to each subcutaneous space between the slips of the plantar aponeurosis. Each is partially subdivided by incomplete transverse septa, particularly at the level of the base of the fifth metatarsal bone where the first and fourth end proximally. The second is the longest and extends posteriorly to within 2 to 3 centimeters of the medial tubercle of the calcaneus. The third is the shortest and runs into the second 2.5 centimeters distal to the base of the fifth metatarsal bone. All four communicate more or less freely, and the septa may be easily broken down. Posterior to these compartments, the aponeurosis and flexor brevis muscle are very closely united and require sharp dissection to separate them. Anteriorly, the compartments end opposite or proximal to the middle of the metatarsal bones where the fascia of the floor and roof come together. This wall may be easily broken through into the subcutaneous interspaces, especially along the digital nerves which are themselves covered by sheaths derived from the fascia of the floor of M1.

MEDIAN PLANTAR SPACE M2

By detaching the plantar aponeurosis and flexor brevis muscle from the calcaneus and turning the resulting flap forward, a second median plantar space, triangular in shape, is uncovered lying between the flexor brevis and the quadratus plantæ muscles, the latter being joined medially by the flexor digitorum longus tendon (Figs. 2, 5, 7). This space is lined with fascia which consists of the sheaths of the flexor brevis and quadratus plantæ muscles and their reflections at the periphery. The apex lies 1 centimeter anteromedial to the medial tubercle of the calcaneus, on, or slightly above, a line between that point and the tuberosity of the navicular bone. Between the quadratus plantæ and its fascial sheath, the lateral plantar nerve and vessels extend in an anterolateral direction, the artery with its two vena comites lying posterolateral to the nerve.

The lateral boundary of M2, which is likewise the boundary of M1 and M3, is a

connective tissue septum continuous superficially with the plantar aponeurosis and extending from the medial tubercle and medial side of the calcaneus to the medial side of the head of the fifth metatarsal bone. The posterior extremity of this septum is fused with the sheath of the abductor digiti quinti and in this region the lateral space deep to that muscle and the median space M2 are separated only by the double septum resulting from this fusion. In the posterior half of the foot, the septum is attached deeply to the tarsal bones and ligaments, and is directed medially as well as posteriorly and deeply as it approaches the calcaneus, so that a portion of the lateral space intervenes between M2 and the calcaneus. Anteriorly, it attaches on its deep side to the inferiomedial border of the third plantar interosseous and, splitting to inclose this muscle, finally gains attachment to the medial border of the fifth metatarsal bone and to the proximal phalanx at the insertion of the muscle. This portion is, therefore, directed deeply and laterally.

Anteriorly, the lateral boundary of M2 and the sheath of the abductor digiti quinti are separated by the lateral plantar structures and the flexor digiti quinti brevis muscle. As the lateral plantar artery crosses the posterior extremity of M2, deep to the fascia covering the quadratus plantæ muscle, it gives off a branch which perforates or passes under the common septum between that and the lateral space. The main trunk passes through the lateral boundary of M2 about half way between the medial tubercle of the calcaneus and the base of the fifth metatarsal bone, and extends forward between that septum and the sheath of the abductor digiti quinti, soon giving off an additional branch to the lateral space. About 2.5 centimeters distal to the base of the fifth metatarsal bone, it passes deep on the medial side of the flexor digiti quinti brevis and, again piercing the lateral boundary of M2 and M3, enters the latter space by crossing the metatarsal bones and interossei muscles. At the lateral border of the adductor hallucis obliquis, it runs deep to that muscle and forms the plantar arch, giving off the four plantar metatarsal arteries. Before going deep, it sends a metatarsal branch to the lateral side of the little toe. Two vena comites accompany the artery and its branches.

The lateral plantar nerve crosses M2, pierces its lateral wall just distal to the vessels, and, opposite the base of the fifth metatarsal bone, divides into superficial and deep branches, the latter of which accompanies the vessels into the depths of the foot. The superficial branch almost immediately subdivides into a digital nerve to the lateral side of the fifth toe and a nerve which winds around the lateral border of the flexor digitorum brevis into M1 and extends forward to supply the adjacent sides of the fourth and fifth toes.

The medial boundary, which is likewise the boundary of M1, M3, and M4, is a fibrous tissue septum continuous superficially with the plantar aponeurosis and including in its posterior part some of the muscle fibers of the flexor digitorum brevis as the latter bends around medially to gain attachment to the calcaneus. It extends anteromedially from the medial side of the calcaneus to the lateral side of the head of the first metatarsal bone. On its deep side, it becomes attached posteriorly to the tarsal bones and ligaments, and anteriorly to the lateral side of the first metatarsal bone, after passing between the adductor hallucis obliquis and the flexor hallucis brevis and helping to form the sheaths of both muscles. Superficially, its anterior extremity is fused with the insertion of the lateral head of the flexor hallucis brevis and with the fibrous sheath of the flexor hallucis longus, thus gaining attachment to the phalanges of the big toe. Opposite the base of the fifth metatarsal bone, the tendon of the flexor digitorum longus crosses over that of the flexor hallucis longus and pierces the medial wall of M2 and M3 to join the quadratus plantæ muscle but, as it does so, the margins of the wall are closely adherent to it, thus completing the separation of the median foot spaces from the space passing behind the medial malleolus into the leg. However, the separating septum is very thin here and easily broken down, as will be shown in the injection experiments.

Posterior to the base of the fifth metatarsal bone the medial wall is fused with the sheath

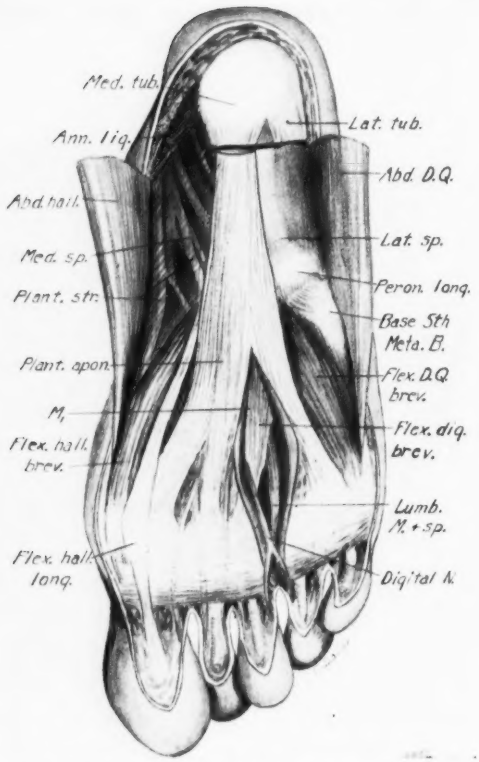


Fig. 1. Superficial plantar view of human foot, showing plantar aponeurosis and related structures.

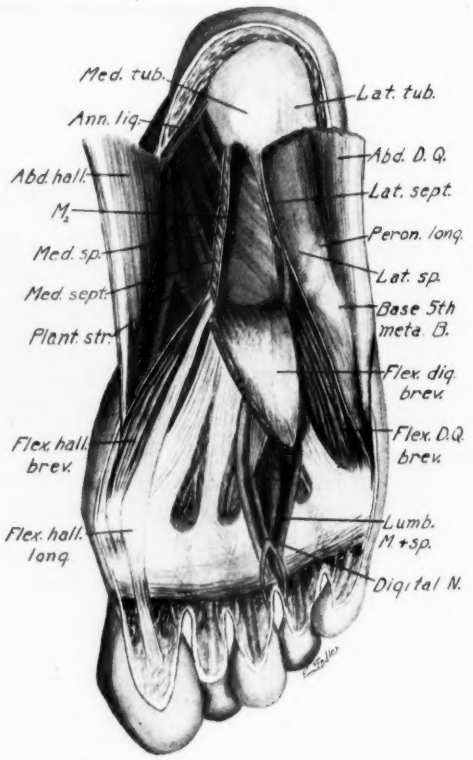


Fig. 2. Same as in Figure 1 but plantar aponeurosis and flexor digitorum brevis muscle reflected anteriorly.

of the abductor hallucis, thus forming a common septum between the medial space deep to that muscle, and M2 in this part of the foot. In the middle of the foot, the medial wall of M2 and the sheath of the abductor hallucis are separated by the crossing of the long flexor tendons. More anteriorly, they are separated by the flexor hallucis longus tendon and the flexor hallucis brevis muscle.

The medial plantar nerve, lying in the medial wall of M2, divides 2 to 3 centimeters anterior to the calcaneus. The medial branch continues forward (medial to M2) and becomes the digital branch to the inner side of the big toe. The lateral branch soon bulges into M2 (though still separated from that space by a layer of fascia), and finally passes around the flexor brevis to enter M1. It then divides into three common digital branches which pass forward superficially to the three medial lumbrical muscles and divide into proper digital branches for the adjacent sides of the corresponding toes. The medial plantar artery (and veins) divides into branches which in general follow the same course as those of the nerve, the medial branch being distributed to the inner side of the big toe and the lateral branch passing into M1 and supplying three common digital branches which anastomose distally with the plantar metatarsal arteries from the deep arch.

Anteriorly, M2 extends as a distinct space only to a line opposite the base of the fifth metatarsal bone where the fascia of the roof and floor are joined by loose areolar tissue. This may be easily broken through and the space extended forward into the interspaces between the slips of the plantar aponeurosis,

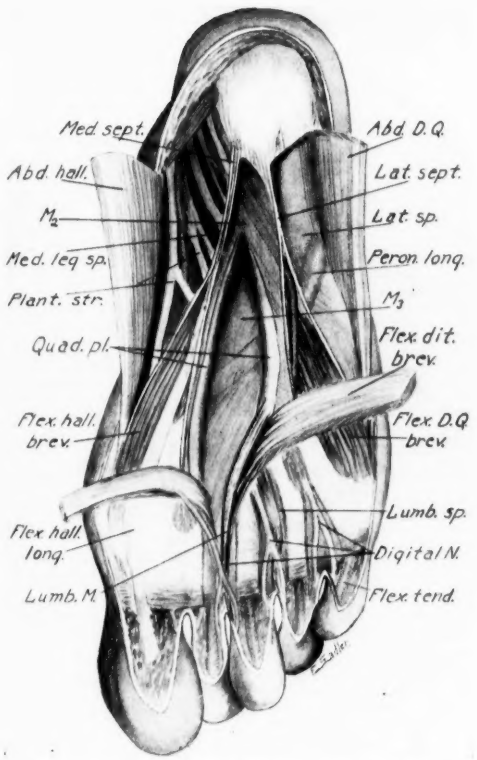


Fig. 3. Same as in Figure 2 but quadratus plantæ muscle incised and spread laterally.

Fig. 4. Same as in Figure 3 but all plantar muscles except the interossei removed.

where it enters superficially to the sheaths of the lumbrical muscles and between them and the overlying fascia (Fig. 7). In breaking through this areolar tissue, it is noted that anteriorly somewhat stronger connective tissue fibers divide this part of M2 into four ill defined compartments, each in line with a lumbrical groove. The walls of these compartments are part of the paratendinous sheaths described later.

MEDIAN PLANTAR SPACE M3

If the quadratus plantæ muscle and its sheath be detached from its origin on the calcaneus and from the tendon of the flexor digitorum longus and turned forward, still another median plantar space (M3) is found deep to it (Figs. 3, 4, 5, 7). This space is also triangular in shape, with the apex lying 2 to 3 centimeters anterior to that of M2. The floor of the space consists of the tarsal bones and ligaments and, more anteriorly, the tendon of the peroneus longus, the adductor hallucis obliquis, and the plantar and dorsal interossei muscles—all covered by fascia. The medial boundary of M3 is continuous with that of M1, M2, and M4. Due to the concavity of the main arch of the foot, the lateral boundary of M3 consists chiefly of the tarsal bones and ligaments (especially the long plantar ligament) posteriorly, and the third plantar and fourth dorsal interossei muscles anteriorly. Since the fibrous tissue lateral boundary of M1 and M2 joins the tarsal bones and ligaments posteriorly and the margin of the third plantar interosseous muscle anteriorly, it likewise comes into relation with M3 superficially. Because of the

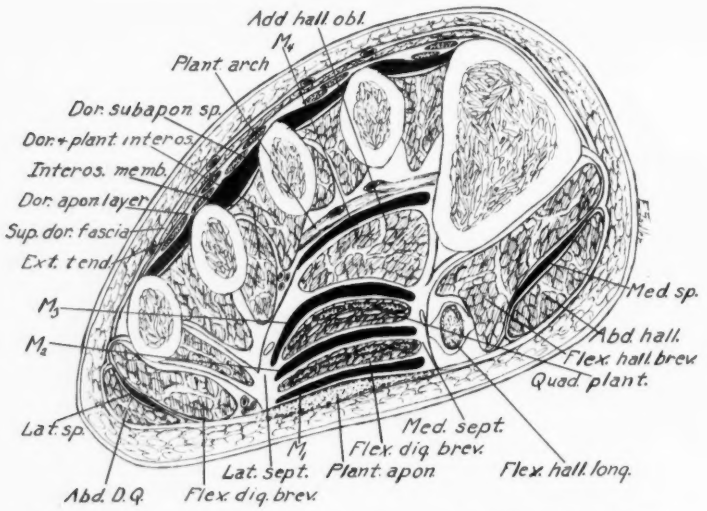


Fig. 5. Cross section of foot at level of the middle of the fifth metatarsal bone (proximal surface).

concavity of the arch, M_3 is deeper and lies farther away from the surface on the medial side of the foot.

There are a few connective tissue fibers between the fascia of the roof and floor of M_3 opposite the base of the fifth metatarsal bone but, on a line between the middle of that and the head of the first metatarsal bone, the anterior septum becomes complete. However, again it consists of loose areolar tissue which may be easily separated into four compartments, thus leading into the lumbrical grooves deep to the muscles and their fascia. Opposite the heads of the metatarsal bones these compartments suddenly narrow down so that M_3 can be traced no farther forward except by breaking through the fascia separating it from the lumbrical muscles and following those muscles. The walls of these compartments are again parts of the paratendinous sheaths.

MEDIAN PLANTAR SPACE M4

This refers to the potential space lying deep to the oblique head of the adductor hallucis muscle which takes origin from the sheath of the peroneus longus tendon and the bases of the second, third, and fourth metatarsal bones, and is inserted into the lateral side of the base of the first phalanx of the big toe (Figs. 4, 5, 7). This muscle overlies all or part of the plantar and dorsal interossei muscles and sheaths in the three medial interspaces. The interossei of the fourth interspace (third plantar and fourth dorsal) are entirely in the clear, while the first dorsal interosseous in the first interspace is almost entirely covered by this head. The proximal half of the second interspace (first plantar and second dorsal interossei) lies under the adductor obliquis and likewise a small portion of the proximal part of the third interspace (second plantar and third dorsal interossei). The first interspace is, therefore, in relation to M4, the fourth and almost all the third to M_3, but the second is in relation to both: proximally to M4 and distally to M_3. The importance of this relationship to the spread of infection into the dorsal subaponeurotic space will be shown in the injection experiments.

Superficial to the adductor obliquis and its sheath, lie the slips of the flexor digitorum longus and the lumbrical muscles enclosed in their sheaths, with M_3 intervening. The medial boundary of M4, which helps form

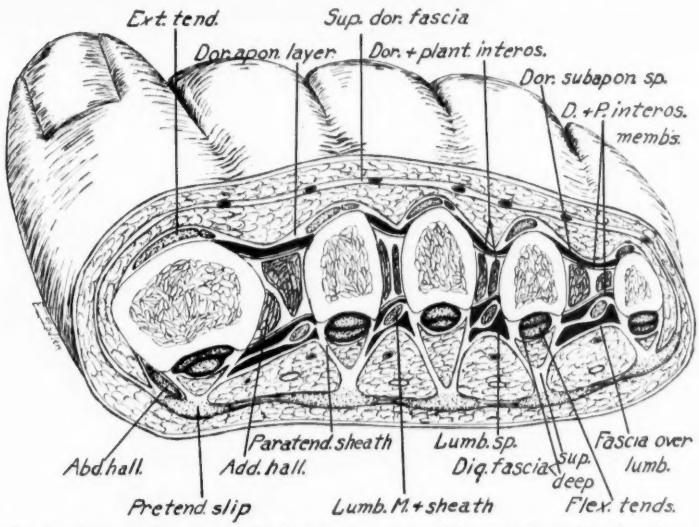


Fig. 6. Cross section just proximal to the metatarsophalangeal joints (distal surface).

the sheath of the adductor obliquis muscle, is on a line with the lateral side of the first metatarsal bone and is continuous with the medial boundary of M1, M2, and M3. The lateral boundary is the sheath of the muscle and is on a line between the base of the fourth metatarsal bone and the lateral side of the base of the first phalanx of the big toe.

The plantar arch of blood vessels lies for the most part deep to the adductor obliquis muscle, and the plantar metatarsal vessels take origin there (M4). The deep branch of the lateral plantar nerve follows the arch across this space.

LUMBRICAL AND SUBCUTANEOUS INTERSPACES

The interspaces between the slips of the plantar aponeurosis are occupied by subcutaneous fatty tissue (superficial fascia) in the substance of which run the common digital nerves, surrounded by their sheaths derived from the fascial floor of M1, and distally also the plantar metatarsal vessels which become superficial near the necks of the metatarsal bones (Figs. 1 to 7). If the nerve and vessel be dissected out and the fatty tissue removed, a definite and fairly firm layer of fascia is seen overlying each lumbrical muscle and extending between the lateral walls of the lumbrical groove. Proximally, the subcutaneous interspaces superficial to this fascia are separated from M1 by transverse connective tissue septa about 3 to 4 centimeters proximal to the heads of the metatarsal bones. These septa are easily broken through, particularly along the digital nerves which pass from M1 into the subcutaneous interspaces. Cutting through the fascia overlying the lumbrical muscles exposes the latter, each enclosed in a thin fascial sheath derived from that of the flexor digitorum longus and quadratus plantæ muscles from which it takes origin on a line between the middle of the fifth and the head of the first metatarsal bones. This sheath is attached to the lateral wall on either side of the groove. At their origins, the lumbrical muscles are in relation with the short flexor slips which cross them superficially and the deep flexors which are lateral to them. Anteriorly, both tendons lie lateral to the lumbrical grooves and are separated from them by their fascial coverings derived from the muscles from which they originate. These coverings correspond to the paratendinous sheaths of the hand described by Kanavel and his associates and are reenforced by deep bands from the plantar

aponeurosis to form the lateral walls of the lumbrical tunnels. Anteriorly, these walls gain attachment to the capsules of the metatarsophalangeal joints, to the transverse metatarsal ligaments, and to the deep layer of the digital sheaths.

The potential space between each of the lumbricals and the overlying fascia is separated from the distal end of M_2 by loose areolar tissue which is easily broken through putting these spaces into communication. Distally, at its insertion into the extensor tendons, each lumbrical extends into the dorsal subcutaneous region. Deep to each lumbrical muscle and its sheath, lies the loose areolar tissue in the corresponding anterior compartment of M_3. This tissue is also easily broken through, thus placing the lumbrical groove into communication with M_3.

DIGITAL SHEATHS

There is a common synovial sheath for the long and short tendons to each of the lateral four toes and one for the long tendon to the big toe. These extend from the insertions of the tendons distally to or beyond the necks of the metatarsal bones proximally. Each synovial sheath is surrounded by a dense fibrous covering, the deep layer of the digital sheath, which binds the tendons to the sides of the phalanges and ends opposite the head of the metatarsal bone, or rather becomes much thinner there and continues proximally, over the tendons and deep to the aponeurosis, to merge with the fascia over the flexor digitorum brevis (Figs. 5, 6). Laterally, this layer continues around the tendons as the paratendinous sheath and, together with the re-enforcing bands from the plantar aponeurosis, forms the lateral wall of the lumbrical groove. Overlying the deep digital sheath is another, less dense layer containing the digital nerves and vessels and receiving proximally part of the digital slip of the plantar aponeurosis. Some of these aponeurotic fibers cross over obliquely from one side of the proximal phalanx to the other, somewhat similar to the condition described in the hand by Kanavel, and help explain the displacements of the digital structures in Dupuytren's contraction.

LATERAL PLANTAR SPACE

This is the potential space under the abductor digiti quinti, from its origin to its insertion, and between that muscle and the deep part of its sheath (Figs. 1, 2, 3, 5, 6). The superficial portion of the sheath is the thickened lateral band (calcanometatarsal band) of the plantar aponeurosis and the deep portion is the fascia overlying the long plantar ligament, the tendon of the peroneus longus muscle as it enters the foot and, more anteriorly, the flexor digiti quinti brevis muscle. Laterally, the superficial and deep portions of the sheath come together to shut off the lateral plantar space from the lateral leg space along the peronei tendons and muscles.

MEDIAL PLANTAR SPACE

This is the potential space deep to the abductor hallucis and between that muscle and its sheath (Figs. 1, 2, 5, 6). It extends from the origin of the muscle on the calcaneus to the point where its tendon fuses with the medial head of the flexor hallucis brevis (2.5 centimeters anterior to the base of the fifth metatarsal bone). The fascial sheath deep to this space covers the posterior tibial and plantar structures as well as the long flexor tendons entering the foot behind the medial malleolus and shuts off those structures from this space. The sheath is completed superficially and medially by the thin medial portion of the plantar aponeurosis.

DORSAL FOOT SPACES

Two distinct spaces can be recognized, subcutaneous and subaponeurotic (Figs. 5, 6, 7). The former is continuous with the subcutaneous regions adjacent and receives the insertions of the lumbrical muscles, just as in the hand. The subaponeurotic space is that lying deep to the deep fascia overlying and enclosing the extensor tendons, and between that fascia and the dorsal interosseous membrane. It is in relation with M_3 and M_4 through the interosseous spaces as described.

MEDIAL LEG SPACE

Following superficially and proximally along the tendons of the flexor hallucis longus, flexor digitorum longus, and tibialis

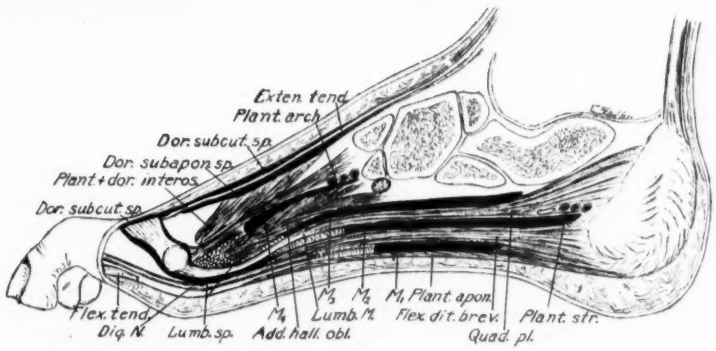


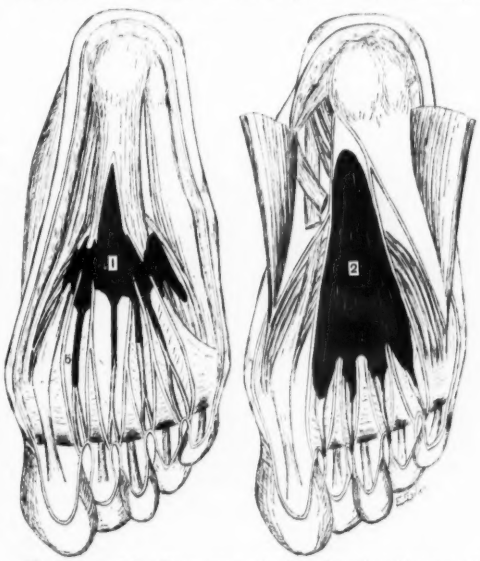
Fig. 7. Sagittal section of foot between the second and third toes (lateral surface).

posterior, one comes to a large potential space (Fig. 15) between those muscles and the superficial calf muscles (gastrocnemius and soleus). The upper extremity of this space corresponds to the origin of the soleus from the tibia and fibula at the junction of the middle and upper thirds. In it lie the posterior tibial vessels and nerves which extend between the origins of the soleus still farther proximally into the popliteal space. Distally these structures pass under the annular ligament toward the medial wall of M2 and M3, dividing meanwhile into medial and lateral plantar branches.

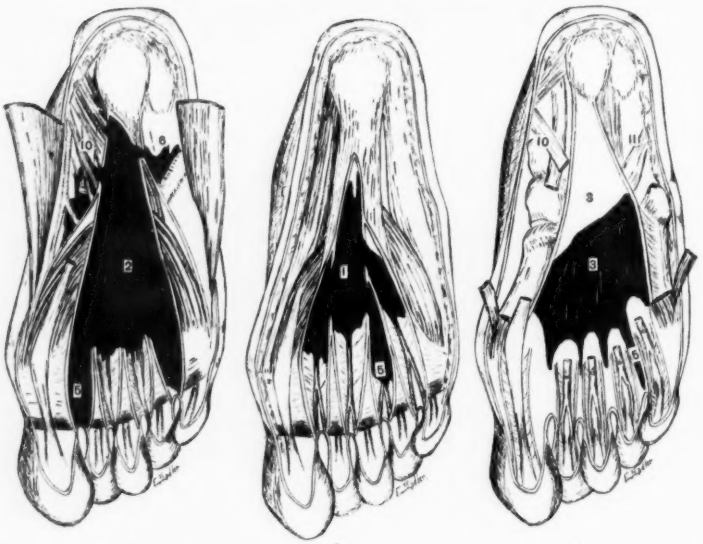
The long flexor tendons likewise lead under the annular ligament and abductor hallucis muscle (but separated from the medial space by a distinct layer of fascia) to the medial side of M2, where the flexor digitorum longus crosses over the flexor hallucis longus and pierces the medial wall to join the quadratus plantæ muscle, while the flexor hallucis longus continues forward and superficially toward the big toe. The tibialis posterior, after having been crossed over by the flexor digitorum longus in the lower third of the leg, lies more anteriorly and passes across the tuberosity of the navicular bone anterolaterally toward its insertion. It is seen, therefore, that, although there is no direct connection between the leg and foot spaces, certain structures (tendons, nerves, and vessels) pass from one to the other and make probable the extension of injection material or infection alongside them. However, the tight annular ligament offers strong resistance to passage of material under it proximally or distally.

LATERAL LEG SPACE

Within the sheaths of the peroneus longus and brevis, a potential space can be traced proximally to the upper third of the leg (Fig. 16). A break through this sheath posteriorly would lead into the large medial leg space between the long flexor and superficial calf muscles. A break anteriorly or superficially



Figs. 8a and b. Diagrammatic drawing showing spread of injection mass from M1. *1*, M1; *2*, M2; *5*, subcutaneous interspace along digital nerve.



a. b. c.

Figs. 9a, b, and c. Diagrammatic drawing showing spread of injection mass from M2. *1*, M1; *2*, M2; *3*, M3; *5*, subcutaneous and lumbrical interspaces; *6*, lateral plantar space; *10*, medial leg space; *11*, lateral leg space.

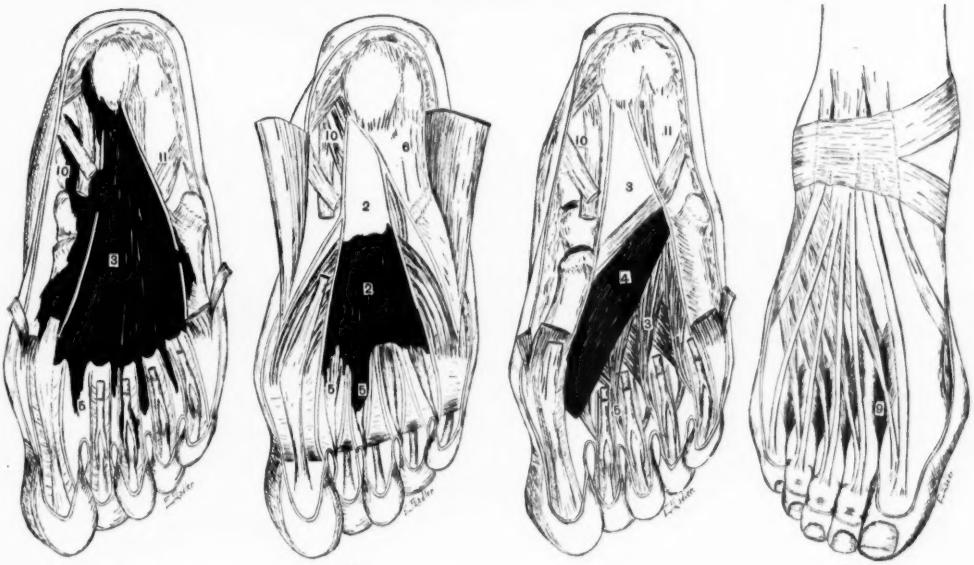
would lead to subcutaneous infiltrations. Distally, after passing under the lateral annular ligament, the tendon of the peroneus brevis bends sharply anteriorly to its insertion on the base of the fifth metatarsal bone while that of the peroneus longus enters the foot behind and deep to the base of the fifth metatarsal bone and passes anteriorly, medially, and deeply to its insertion on the first cuneiform and first metatarsal bones. In its course under the abductor digiti quinti, the peroneus longus is separated from the lateral plantar space by a definite and heavy layer of fascia as well as by its own fibrous sheath. In the depths of the foot, it is separated from M3 and M4 by its sheath. The lateral leg space is, therefore, also separated from the foot spaces but again we may anticipate rupture into the foot, particularly into the lateral plantar space and the median plantar spaces M3 and M4 because of their close relationship to the peroneus longus tendon.

INJECTION EXPERIMENTS

In a total of 58 injections, two types of material were used: paraffin colored with Sudan III or spirit blue, and gelatin colored with India ink. The latter was found to be most satisfactory because of the slower setting time and ease of handling. Both fresh and preserved human material were used. A few injections were made under pressure bottle control but most were done with a Luer syringe and needle, the pressure being controlled by the volume used. Attempts were made to fill the various spaces found in the dissections. In some cases, these masses were confined to the spaces injected but in others they spread into adjacent spaces.

Extension from M1 (*Fig. 8*). All the injections into this space showed breaks medially and laterally, as well as through the plantar surface of the aponeurosis. In all, the mass had extended beyond the anterior extremity of M1 along the digital nerves into the subcutaneous interspaces. Likewise, in all, the mass had extended between and around the slips of the flexor digitorum brevis into M2, whence the usual spread took place.

Extension from M2 (*Fig. 9*). In most of the specimens, the mass in M2 lay deep to the fascia covering the quadratus plantæ muscle.

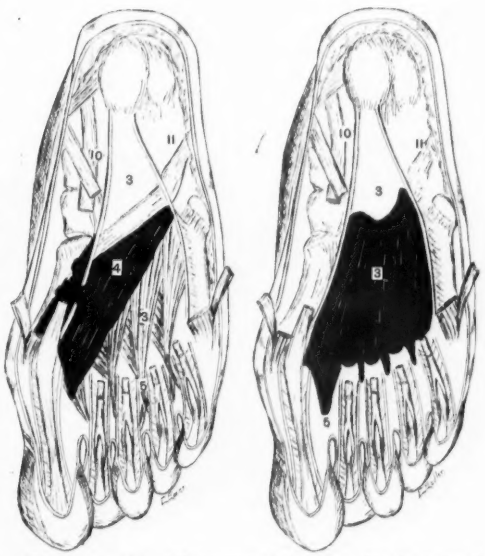


a. b. c. d.

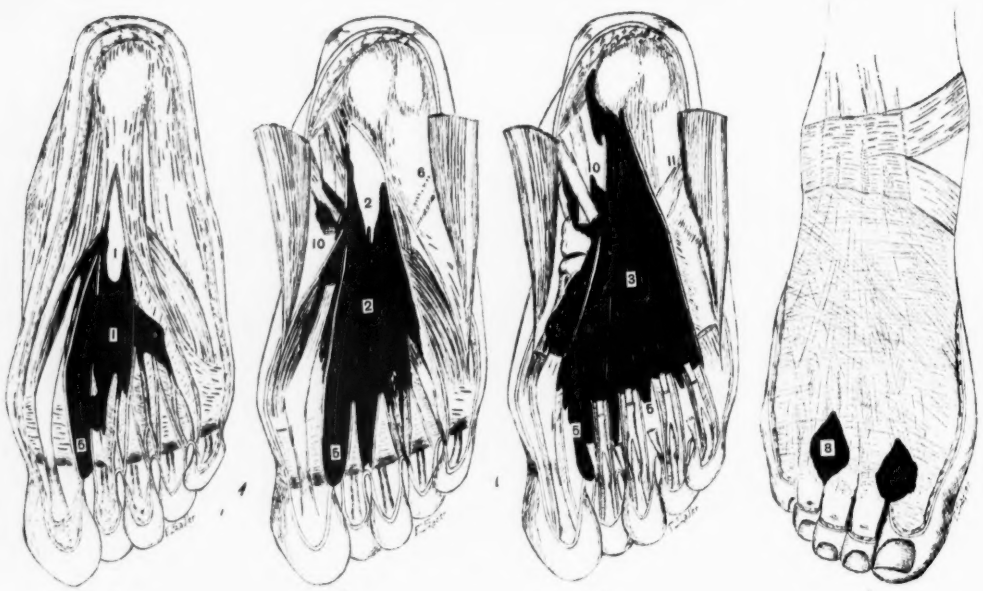
Figs. 10a, b, c, and d. Diagrammatic drawing showing spread of injection mass from M3. *2*, M2; *3*, M3; *4*, M4; *5*, lumbrical spaces; *6*, lateral plantar space; *9*, dorsal subaponeurotic space; *10*, medial, and *11*, lateral leg space.

The most common extensions from this space were into M3 (100 per cent), into the lumbrical spaces (100 per cent), and into M1 (75 per cent). In no case did the spread into M3 go posterior to the base of the fifth metatarsal bone. It occurred anteriorly between and around the slips of the quadratus plantæ muscle. Along the lumbrical muscles, the mass had spread a variable distance—in some cases to their insertions dorsally. In some, the mass was confined within the sheaths of the muscles or more commonly between those sheaths and the overlying fascial coverings, but in others it had broken through to lie more superficially. There were two possible routes of extension from M2 into M1; one by first spreading to the lumbrical grooves and from there along the subcutaneous interspaces into M1, and the other by breaking through and around the slips of the flexor digitorum brevis directly into M1. There was evidence of spread by both routes in our specimens.

In 50 per cent, there was an extension through the medial wall to the crossing of the long flexor tendons, with more or less spread



Figs. 11a and b. Diagrammatic drawing showing spread of injection mass from M4. *3*, M3; *4*, M4; *5*, lumbrical space; *10*, medial leg space; *11*, lateral leg space.



Figs. 12a, b, c, and d. Diagrammatic drawing showing spread of injection mass from lumbrical spaces. *1*, M1; *2*, M2; *3*, M3; *5*, lumbrical spaces; *6*, lateral plantar space; *8*, dorsal subcutaneous space; *10*, medial leg space; *11*, lateral leg space.

from there proximally along both tendons and distally along that of the flexor hallucis longus. In about an equal number, there was a spread proximally along the lateral plantar structures, as far as or above the annular ligament. Less frequent extensions from M2 were proximally along the medial plantar structures (25 per cent) and laterally into the space under the abductor digiti quinti (25 per cent). The latter apparently took place along the vessels perforating the common septum between those spaces.

Extension from M3 (*Fig. 10*). The most common extensions were through the medial wall to the crossing of the long flexor tendons (90 per cent), into M2 (75 per cent), and into the lumbrical spaces (75 per cent). In some the spread to the long flexor tendons was confined to the area of crossing, but in others the mass extended proximally along both tendons to, or even beyond, the annular ligament and distally along the flexor hallucis longus tendon toward its insertion. In one specimen, the injection had broken into the synovial sheaths of the long flexors and had extended to their upper limits above the malleolus. The spread into M2 varied in amount, but in every case had occurred anteriorly between and around the deep flexor slips. In the extensions into the lumbrical spaces, the mass lay deep to the sheaths of the muscles in some but in others had broken through them and the overlying fascia to become more superficial. Distally, the lumbrical masses passed a variable distance—even to the insertions of the muscles dorsally.

About 50 per cent of the injections extended into M1, either directly or secondarily from M2 or the lumbrical grooves. There was direct extension into M4 in 40 per cent and in an equal number there was a break through the posterior medial extremity of M3 to the medial side of the calcaneus, deep to the posterior tibial and lateral plantar structures but separated from them by a definite layer of fascia. Another 40 per cent showed spread through the distal half of the second and all of the third and fourth interosseous

spaces into the dorsal subaponeurotic space. Less frequent was the spread along the deep side of the flexors to the toes to, or almost to, the metatarsophalangeal joints. In one specimen, the mass had broken into the synovial sheath of the third toe, extending to its distal end. Other infrequent extensions were through the lateral wall to the deep side of the flexor digiti quinti brevis and through the medial wall to the deep side of the flexor hallucis brevis muscle.

Extension from M4 (*Fig. 11*). In every case there was extension into M3. This was to be expected from the small size of M4 and its close anatomical relationship to M3. In about half of the cases, the mass broke through the first and proximal half of the second interosseous spaces to reach the dorsal subaponeurotic space. In 30 per cent, there was extension through the medial wall to the deep side of the flexor hallucis brevis muscle.

Extension from lumbrical spaces (*Fig. 12*). About 90 per cent of these injections showed breaks superficially into the subcutaneous portions of the lumbrical tunnels, from which the mass in most cases extended into M1 or superficially over the plantar aponeurosis or both. Almost as many (80 per cent) showed extension into the dorsal subcutaneous space at the insertions of the muscles. Extension into M2 superficially to muscles and into M3 deep to them occurred in 70 per cent.

Extension from lateral plantar space (*Fig. 13*). The most common spread from this space was through its medial and lateral walls to the subcutaneous regions adjacent (100 per cent). In 66 per cent, there was extension into M2 through the common septum between, the first break occurring along the vessels piercing that septum. In a like number, there was spread through the floor or lateral wall to peroneus longus tendon, then proximally and distally along this tendon.

Extension from medial plantar space (*Fig. 14*). This space was quite independent of the other foot spaces. The only consistent spread from it was through its medial and lateral walls subcutaneously.

Extension from region behind medial malleolus (*Fig. 15*). The most common route of extension from the region deep to the deep

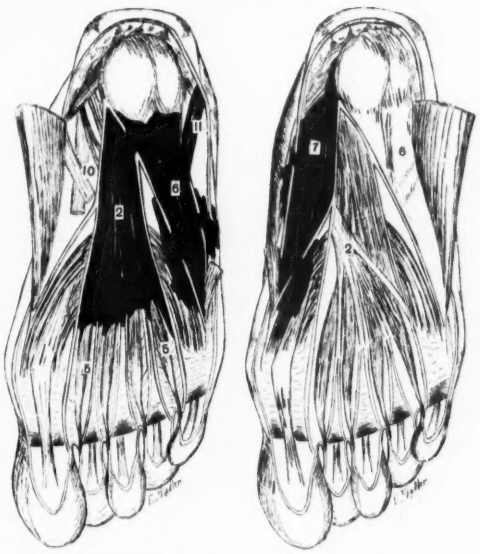


Fig. 13 (left). Diagrammatic drawing showing spread of injection mass from lateral plantar space. *2*, M2; *5*, lumbrical spaces; *6*, lateral plantar space; *10*, medial leg space; *11*, lateral leg space.

Fig. 14. Diagrammatic drawing showing spread of injection mass from medial plantar space. *2*, M2; *6*, lateral plantar space; *7*, medial plantar space.

fascia behind the medial malleolus was proximally into the intermuscular space between the superficial and deep calf muscles and even farther, along the posterior tibial and the popliteal structures (100 per cent). Distally the mass was most apt to go deep to the medial space under the abductor hallucis muscle along the lateral plantar structures into M2 (80 per cent), along the medial plantar structures to the subcutaneous region medial to M2 (80 per cent), along the flexor hallucis longus (80 per cent) and flexor digitorum longus (40 per cent) tendons for a variable distance, and finally from the medial side of the calcaneus into M3 (20 per cent). When the synovial sheaths of the long flexors were entered, they ruptured proximally and distally, with increasing pressure, and the mass took the course described.

Extension from region behind lateral malleolus (*Fig. 16*). The injections into the synovial sheaths of the peronei were either localized within those sheaths or broke through proximally to the upper third of the leg or distally

Fig. 15b.

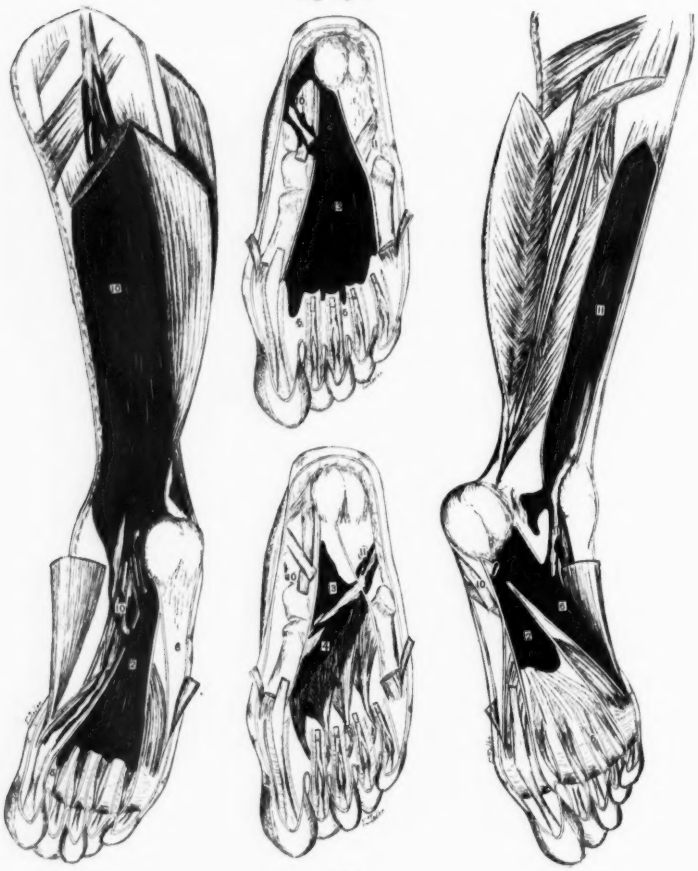


Fig. 15a. Fig. 16b. Fig. 16a.

Figs. 15a and b. Diagrammatic drawing showing spread of injection mass from region behind medial malleolus. *2*, M2; *3*, M3; *5*, lumbrical spaces; *6*, lateral plantar space; *10*, medial leg space; *11*, lateral leg space.

Figs. 16a and b. Diagrammatic drawing showing spread of injection mass from region behind lateral malleolus. *2*, M2; *3*, M3; *4*, M4; *5*, lumbrical spaces; *6*, lateral plantar space; *10*, medial leg space; *11*, lateral leg space.

into the foot. The latter extension was most often into the lateral space under the abductor digiti quinti from which further spread into M2 was common. Spread also occurred subcutaneously at the lateral border of the abductor or deeply along the peroneus longus tendon into the depths of the foot from which location the mass often went into M3 and M4. The injections, superficial to the synovial sheath but deep to the deep fascia, traveled up the leg within the muscle sheaths rather than distally into the foot—the resistance to the flow apparently being less upward.

INCISIONS FOR DRAINING FASCIAL SPACES

The above study of dissections and injections of the fascial spaces has suggested some anatomical methods of approach for draining infections of these spaces. M1 may be opened from the plantar side but, although this is

the most logical approach, it has the objection of leaving a scar on the sensitive plantar surface of the foot which may be a source of discomfort later. In addition, one must keep in mind that septa divide M1 into several compartments and that these septa must be broken down to open up all pockets of pus.

M2 may also be opened from the plantar side, but this procedure is open to the same objection as that for M1 and in addition necessitates going through the flexor brevis muscle, with resulting poor drainage and later possible loss of function. The plantar approach to M3 and M4 is open to all the above objections, and in addition there is the danger of injury to the lateral plantar structures crossing the floor of M2. A much better method of approach to M2, M3, and M4 is from the medial side. A study of Figure 5 shows that all of these spaces have a common medial wall which separates them from the flexor hallucis longus and brevis and more medially from the abductor hallucis muscles. An incision on the medial side of the foot, opposite the anterior surface of the first metatarsal bone, leads under the abductor hallucis and flexor hallucis brevis muscles to the common medial wall from which any of the median plantar spaces may be easily entered by pushing a hæmostat through at the proper level. Since infection is apt to spread from one to the other of the median spaces, a method of approach that gives access to all of them is doubly valuable. Counter drainage may be obtained by advancing the hæmostat to the lateral wall of the space opened into and from there to the plantar surface at the superficial margin of the third plantar interosseous muscle to which the lateral wall is attached. Counter drainage from the lateral side along the anterior surface of the fifth metatarsal bone is interfered with by the superficial position of the third plantar and fourth dorsal interossei which would have to be cut through in such an approach. The opening medially into M3 may be extended posteriorly by following the tendon of the flexor hallucis longus to its crossing by the flexor digitorum longus and entering M3 deep to that crossing. M2 may likewise be entered superficial to the crossing.

In an infection of a lumbrical space, the latter should be opened, just as in the hand, from the medial side of the proximal phalanx to the proximal end of the space (on a line between the middle of the fifth and the head of the first metatarsal bones). If there has been extension to the dorsal subcutaneous space, the incision should likewise be extended dorsally from its distal end. As most lumbrical space infections are accompanied by involvement of the median plantar spaces, it may be necessary to open into the latter from the proximal end of the lumbrical incision: a hæmostat directed proximally deep to the lumbrical muscle entering M3, superficial to it M2, and along the digital nerve M1. For additional drainage, the median spaces should be opened from medial side as stated.

Infections in the lateral space under the abductor digiti quinti are best drained through the lateral wall of that space and those in the medial space under the abductor hallucis through the medial wall. These are easily taken care of but if spread occurs to other spaces, the latter must be opened in the manner described for them.

Dorsal subaponeurotic infections are apt to remain localized and are easily opened from the dorsum of the foot, but if they are secondary to infections in M3 or M4, through and through drainage must be instituted.

Infections in the deep leg space between the superficial and deep calf muscles are best drained by incisions on both sides of the tendo achillis extended proximally along the borders of the gastrocnemius and soleus to the upper third of the leg. Through and through drainage may be established. On the medial side, the incision may be extended distally behind the malleolus along the long flexor tendons and posterior tibial structures.

Infections along the peronei should be opened directly over those muscles and the incisions extended proximally or distally as indicated.

REFERENCES

1. KANAVEL, ALLEN B. Infections of the Hand. 4th ed. Philadelphia and New York: Lea & Febiger, 1921.
2. KANAVEL, ALLEN B., KOCH, SUMNER L., and MASON, MICHAEL L. Dupuytren's contraction. Surg., Gynec. & Obst., 1929, xlviii, 145.

THE POSTURAL TREATMENT OF POSTOPERATIVE MASSIVE ATELECTATIC COLLAPSE[1]

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THOUGH postoperative massive atelectatic collapse of the lung has been accepted by the profession as a clinical entity, the etiological factors producing the condition, its mode of production, and by what methods it can best be terminated once it has occurred are problems which are still under discussion. Our data is derived for the most part from the study of 14 cases which have occurred at this hospital during the years 1925 to 1928 inclusive. The pathological data was obtained from the necropsy reports of Bergamini and Shepard and Pasteur's description of his fatal cases, as none of our cases have terminated fatally during the presence of the disease.

We thought it would not be amiss to review the disease as we have encountered it in this relatively large group of cases, so that the process might be more clearly outlined clinically.

Definition. Postoperative massive atelectasis of the lung is a reaction of an obstructive nature in which the affected part of the lung becomes airless, characterized by displacement of the mediastinal contents to the involved side, decreased radiability of the affected lung tissue, a high diaphragm and flattened chest on the same side, increased respiratory and pulse rate, cyanosis, and usually a rather abrupt onset within 48 hours after an operation or injury.

History. W. Pasteur (18) in 1890 first described the condition from a study of 34 cases; he supplemented this in 1908 with his Bradshaw Lecture (19) and in 1914 with another paper (20). Since then a number of excellent articles have been written on the subject, especially those of Bradford, Scott (28), and Sante. Of interest is the fact that most of the authors believed the condition to be neurogenic in origin, but it now appears that the pendulum is swinging toward the theory that obstruction causes the condition, as was pointed out first by Elliott and Dingley, and later by Chevalier Jackson and Walter L. Lee.

Sex. Males and females are equally liable to the disease; in our series of 14 cases the sexes were equally divided.

Age. Age seems to play but little part in the condition in our group. The ages ranged from 12 years to 64 years. However, 6 of the cases were in the third decade.

Season. Ten of the cases occurred in the first 3 months of the year, March alone having 6 to its credit, and it would seem from this that there is a seasonal variation in the incidence of the disease.

The type of operation seemed to play no part in its promotion as there were three cholecystectomies, two gastric operations, four pelvic operations, four appendectomies, and one exploratory laparotomy. It is interesting to note that in a series of 2,346 operations performed at this clinic on patients with intracranial neoplasm, no case of massive atelectatic collapse of the lung has been noted.

Anæsthesia. Eleven patients received ether alone and the remaining three had nitrous oxide gas and oxygen supplemented by ether.

Lung affected. In 13 cases the right lung was involved, and in 10 the right lower lobe was affected. The whole left lung except the extreme apex was involved in the remaining case.

Mode of production. We believe that collapse of the lung is primarily due to obstruction of a bronchus or several bronchi by tenacious mucus and not by a mucus plug. This, we believe, accounts for the absence of postmortem findings demonstrating an actual obstruction, as a foreign body type of mucus plug has usually been expected. This obstructing material is of primary importance; however, there are several secondary factors which greatly facilitate the collapsing of pulmonary tissue, namely, limited excursion of

[1] Read before the Harvard Medical Society, January 22, 1929.

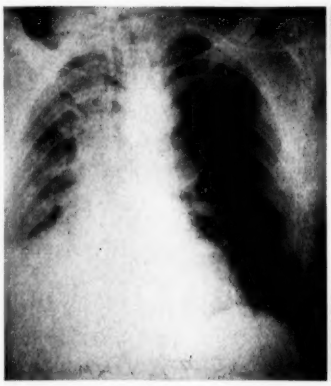

Fig. 1.

Fig. 2.

Fig. 3.

Fig. 1. Case 1. Film taken at 10 o'clcock showing diffuse cloudiness of the whole right lung with consolidation of the lower lobe, displacement of the heart and trachea to the involved side, and a flattening of the right chest. The right diaphragm is obscured.

Fig. 2. Case 1. Showing definite clearing of the right lung. The trachea has returned to its normal position, the heart is still slightly displaced to the right, and the right diaphragm is still slightly higher than normal, but is now visible through the partly aerated lower lobe.

Fig. 3. Case 1. This film 24 hours after the onset of the disease shows only a slight residue of the atelectasis in the right base. The mediastinal contents are in their normal position, but the right diaphragm remains slightly elevated.

the diaphragm and thorax, the habitus of the patient during or following operation, both causing a decreased aeration of the lungs, and probably a raised threshold value of the cough reflex favoring stagnation and drainage of the secretions into the dependent lung, for if the patient occupies the supine position, the right lung will be the one affected in at least four of five cases because of the anatomical structure of its primary bronchus.

That the condition does not occur contralateral to the side operated on, unless the contralateral side is dependent, we feel is shown by our series of cases in which atelectasis developed on the right side in all but one case, even though there were three McBurney incisions, eight right rectus incisions, and three in the midline. Further, the fact that the right lung and especially the right lower lobe are so much more frequently affected than the left forces us to dismiss the theory of a predisposed pulmonary nervous mechanism as of primary importance.

As to the site at which de-aeration commences, we feel that in the milder cases the obstructing material is only sufficiently great to close off the smaller bronchioles, but from this point the process may extend to the larger bronchi; or the original site of obstruction with tenacious mucus may be in one or more of the larger bronchi, with a consequent involvement of a larger area of the lung. Following the obstruction, the air in that part of the lung tissue, the bronchus of which is obstructed, is absorbed by the pulmonary circulation. The absorption of the gases tends toward a negative pressure in the tissues involved and makes conditions optimum for secretion from the cells lining the bronchioles, and also permits or assists fluid to permeate the capillary walls and fill the alveolar cells, which we believe accounts for the heavy congested lungs and hydropic alveolar cells which have been found at postmortem.

That the first step in collapse of the lung after the obstruction has occurred is the absorption of air from the alveoli was demonstrated in one of our cases. The first X-ray film showed the mediastinal structures to be displaced, a high diaphragm was present, and the chest wall appeared retracted, but there was no increased density of the lung. Subsequently, however, the lobe became less radiable and the picture was complete.

Conversely, when the patient is treated the chest structures do not return to normal positions until secretion has been coughed up, and only then does lung become more transparent.

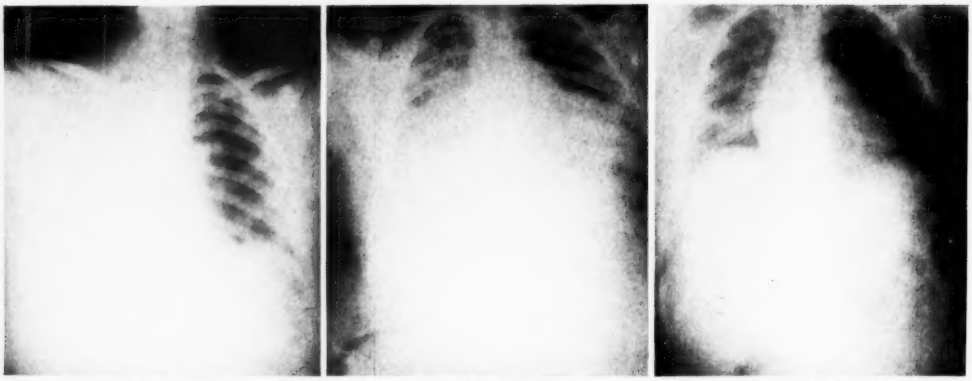

Fig. 4. Fig. 5. Fig. 6.

Fig. 4. Case 2. A film taken at 4 p.m. shows a marked clouding with involvement of right lung except extreme apex laterally. Trachea and heart are displaced to affected side.

Fig. 5. Case 2. Shows a marked clearing of the right lung, while the heart and trachea have returned almost to the midline. However, there is still slight clouding around the right hilus.

Fig. 6. Case 2. Showing further clearing of the right lung with but very slight displacement of the heart and trachea to the right.

Morbid anatomy. Grossly, the affected lung tissue appears smaller than normal, congested, grayish in color, and sinks in water. The lung feels solid, is airless, and does not crepitate. Thick tenacious mucus is frequently found in the bronchi.

Microscopically, the alveolar epithelial cells appear closely packed together and are swollen and hydropic. The capillaries are dilated and congested. The smaller bronchioles are collapsed while the larger ones remain of normal caliber.

Symptoms. The disease usually occurs within 48 hours after operation, although it may occur as late as 6 days following operation. The onset clinically may be abrupt or more gradual, depending on the site of obstruction, whether involving a large bronchus or one of the smaller bronchi respectively. There is a rise in the temperature, although usually only moderate. The respiratory rate is increased and appears to bear a definite relation to the rapidity of onset as it varies directly with the abruptness of onset in many cases. The pulse rate is also increased and depends considerably on the preceding two factors. The patient has no pain, he lies by preference on the affected side, and his breathing is labored, hasty, and jerky. The alæ nasi may dilate with inspiration. In the cases with abrupt onset cyanosis is usually present. Cough is usually absent, although several of our patients have complained of a sensation of "something beneath the sternum which they could not raise." Expectoration is slight, if any, during the actual tenure of the disease, but is great in amount, as is the cough, when the secretion is being expelled. The expectoration, however, is not blood-tinged.

Physical signs. The patient usually lies on the affected side or at least inclines toward it. In the severe cases there is marked respiratory distress, i.e., cyanosis, polyapnœa and hyperapnœa, and the patient looks extremely ill. The respiratory rate ordinarily is around 40 per minute, although in one of our cases the rate was 60 per minute. Movements of the alæ nasi are frequently seen. When only a small portion of the lung tissue is collapsed there is no appreciable change in the size or motion of the two sides of the chest. However, when one or more lobes are involved, there is a definite decrease in the expansion of the involved side and a compensatory increase on the normal side. The point of maximum intensity of the heart beat is displaced toward the involved side.

Palpation confirms inspection as to the decrease in expansion of the diseased lung and also to more accurate localization of the

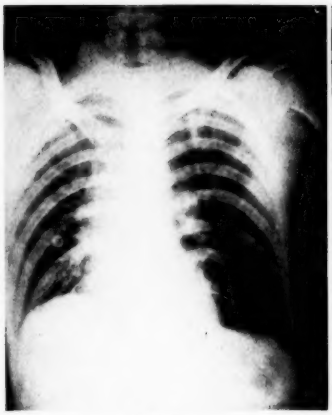

Fig. 7.

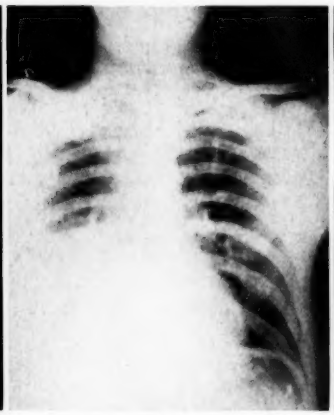

Fig. 8.

Fig. 9.

Fig. 7. Case 3. Showing an early collapse with definite displacement of the mediastinal structures, flattening of chest wall, and beginning de-aeration of right lung.

Fig. 8. Case 3. Re-examination of chest (portable) shows clouding of the right base obscuring diaphragm. The heart and trachea are nearer midline than before. This may be due to exudate in the atelectatic lower lobe.

Fig. 9. Case 3. Third examination of the chest (portable) shows a mottled clouding of the right base, but much less than at last examination. The diaphragm still remains elevated, but the heart and trachea have returned to their normal position. This marked clearing is probably due to postural drainage of the massive atelectasis.

maximum impulse of the heart beat. A friction rub may be felt. Tactile fremitus may be decreased or unaffected, depending on whether the obstructive mechanism predominates or whether the signs transmitted from neighboring patent bronchi predominate.

Percussion confirms palpation as to the location of the point of maximum impulse of the heart. The percussion note may be either dull or flat on the affected side, while hyperresonance of the normal lung is common. The diaphragm on the involved side is high and immobile (Litten's sign is positive).

Auscultation confirms the position of the heart. The auscultatory findings in the lungs may be divided into the following types:

1. Signs of obstruction—the breath sounds are suppressed or merely diminished in intensity. Associated with this we find a decrease in the intensity or absence of whispered and spoken voice. Again in this form few fine râles, if any, are heard.

2. Signs of consolidation—in this form we get tubular breathing and increased whispered and spoken voice sounds. Râles are numerous and vary from the fine crepitant râle to the harsh piping râle so commonly heard in asthmatics.

Further, the above findings are elicited during the onset or actual duration of the disease. However, when the obstruction has been relieved and the patient is coughing up a good deal of mucoid material, râles of all types are heard, and harsh breathing is found. The physical signs may change rapidly, which alone might suggest this condition.

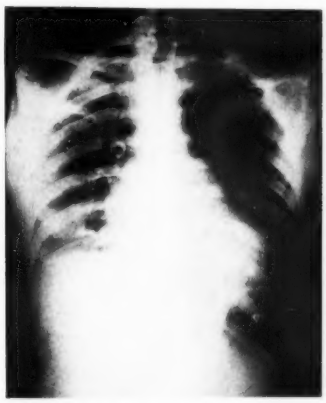

Fig. 10. Case 3. Fourth examination of chest (portable) shows definite clearing in the right base. There is now only moderate increase in the markings to the right base along the heart. The latter is apparently in normal position.

Complications. Only one of our cases developed a complication of a pulmonary nature (interlobar pleurisy), and we are of the opinion that they are very infrequent. However, the occasional finding at autopsy of a completely airless, shrunken, and fibrosed lobe (Mallory[1]) may indicate the possible complication of an organized atelectasis.

Relapse. One patient (Case 5) developed a slight atelectasis on the opposite side. He had been instructed to lie with the left side down and several days later developed a slight atelectasis of the left lower lobe.

Another patient (Case 4) had a recurrence of the process in the same lung three times in the space of 6 days during her illness. It should be stated, however, that the process did not clear completely between the apparent relapses, but did so only partially.

Clinical varieties. These are two in number: (1) Malignant or fulminating type. In this form a patient who is convalescing satisfactorily suddenly becomes cyanotic, has marked dyspnœa, and appears *in extremis*. The respirations are around 60 per minute, pulse is small and rapid, and at times it may reach 140 or more. (2) Insidious. This is the usual mode of onset. In this form a day or two after operation it is noted that the patient is not doing as well as he should. The temperature either remains or becomes elevated, the breathing is somewhat more rapid than usual, and examination demonstrates signs of intrapulmonary changes, often rapidly changing in type. Sometimes the condition is not recognized until cough attracts the physician's attention to the chest. From the study of our cases the diagnosis of atelectasis is seldom made from the physical examination alone.

Prognosis. Postoperative massive atelectasis is rarely fatal in itself and deaths should occur only in the simultaneous bilateral cases, if such may occur. With our present knowledge of the disease practically all clinical evidence of the condition should be absent in 48 hours when the proper treatment is employed. An elevated diaphragm and slight displacement of the mediastinal structures may persist for a number of days but is of little significance.

[1] Personal communication.

Diagnosis. Probably no acute pulmonary affection is so frequently missed as the disease under discussion. It seems that the condition is not thought of and therefore not diagnosed in a large majority of the cases occurring today. To decrease the number of cases undiagnosed or wrongly diagnosed, it might be well to keep in mind the following maxim: "Any postoperative patient that develops pulmonary symptoms and signs within 36 hours after operation should be considered as a case of massive collapse until proven otherwise."

The direct diagnosis is made on finding a displacement of the mediastinal contents to the diseased side, findings consistent with consolidation of the lung, a high diaphragm on the same side, and decreased size and mobility of the chest affected.

Roentgenograms of the chest will demonstrate such findings more clearly than physical examination, for the latter is seldom sufficient except in the cases in which marked atelectasis is present. X-ray films of the chest are indispensable in the diagnosis of minor degrees of atelectasis. The disease is most commonly confused with bronchopneumonia, from which it can be differentiated by the lack of displacement of the mediastinal contents.

Simultaneous bilateral postoperative collapse of the lungs has never been diagnosed in the living as far as we know, and its occurrence theoretically would be incompatible with life. However, marked elevation of both diaphragms is frequently seen in chest films of postoperative patients but without the deaeration of the lungs and displacement of the mediastinum. The consequent diminution of available lung space may force hyperventilation and thus prevent the obstruction of bronchi or bronchioles and typical atelectatic collapse.

Prophylaxis. There are several procedures which appear to be worthy of routine use. (1) Hyperventilation of the lungs during and after operation with carbon dioxide and oxygen as advocated by Scott and Cutler who have demonstrated that by so doing the incidence of massive collapse is diminished by three-fourths; (2) change of position of patient every 6 hours after operation to prevent ac-

Fig. 11.

Fig. 11. Case 4. Four days after operation. This film shows opacity of the left chest except for a small clear area at the apex. The heart is displaced to the left, the diaphragm is high, and the ribs appear retracted.

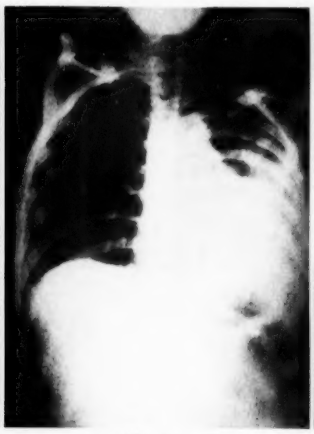

Fig. 12.

Fig. 12. Case 4. Slight spontaneous clearing of the left upper lobe.

Fig. 13.

Fig. 13. Case 4. Recurrence of atelectasis, now almost complete in the left lung.

cumulation or stagnation of secretions in the dependent portions of the lungs; (3) curtailment of postoperative sedatives, especially those which depress the cough reflex and thereby favor stagnation of secretions.

Treatment. The active treatment consists of two procedures: (1) postural and (2) bronchoscopic.

The postural treatment, as first advocated by Sante (25), is simple, safe, and can be carried out by anyone, anywhere. It consists in turning the patient so that the sound lung is dependent, the patient being in a horizontal or slight Trendelenburg position, the latter in case either of the lower lobes are involved. The patient should be kept in this position for a short time, encouraged to cough gently and to breathe as deeply as possible. Gently rolling the patient from side to side or light manual percussion over the involved area may be of considerable assistance in raising mucus. This procedure may be repeated at intervals as often as is necessary, but the patient should not be kept in this position for any length of time as the disease may shift to the dependent lung. This apparently happened in our Case 5.

Five cases which it has been our privilege to observe and treat by postural drainage are reported in detail and the roentgenograms accompany the cases. Brief abstracts of 10 other cases that have occurred in this hospital since 1925 are also recorded.

CASE 1. J. F., Surg. No. 29634, Med. No. 30711. A white married male of 60 entered the hospital on September 13, 1927, for cholecystectomy, which was

Fig. 14. Case 4. Film of the chest at discharge, 2½ months after operation and atelectasis. Complete restoration to normal, but fluoroscopy showed limited movements of the left diaphragm.

performed the following day, under novocain and ether anæsthesia. For the next 2 days the temperature fluctuated around 99, pulse 100, and respirations around 25 per minute. On September 16 the temperature went to 101.6 degrees maximum, the pulse varied from 84 to 120, respirations from 30 to 40 per minute. Examination of the chest at 9 a.m. showed a marked diminution of respiratory movement on the right. The breath sounds were absent over the entire right chest, with bronchial breathing over the left chest. The percussion note of the right chest was dull. The left chest was hyperresonant. Spoken voice was transmitted, but diminished, over the right chest. A few fine râles were heard at the left base. The heart was markedly displaced to the right, both by percussion and by auscultation of the heart sounds. The patient had a chest film taken at this time (Fig. 1).

The patient was returned to the ward and instructed to lie on his left side, and the right side was firmly percussed. Within 30 minutes he began to cough violently and he raised about 100 cubic centimeters of thick, tenacious, mucopurulent sputum. During the next 4 hours he continued to raise lesser quantities of sputum. At 1 o'clock the patient felt markedly improved. At 2:30 p.m. auscultation revealed breath sounds coming through over the right chest and numerous crackling râles were heard. At 4 o'clock the patient was returned to the X-ray room, where another film of the chest was taken (Fig. 2).

At 8 o'clock the temperature had dropped to 99.5 degrees, pulse to 84, and respirations to 30, and the patient was resting quite comfortably.

On the following day, September 17, a third examination of the chest was made (Fig. 3).

The patient's temperature and pulse became normal from this date on, but his respiratory rate remained elevated (around 30) for another day.

On September 28 another film was taken, which showed complete clearing of the lungs. There remained slight bronchial thickening in the right base, and fluoroscopy showed some limitation of the right diaphragm.

The patient was discharged free of symptoms on September 29, 1927, 15 days after operation.

CASE 2. N. I. S., Surg. No. 28334. The patient was a white boy of 12 who entered the hospital on March 6, 1927, complaining of pain in the abdomen. A diagnosis of subacute appendicitis was made, and on March 7, 1927, under a combined anæsthesia of gas and ether, an appendectomy was performed through a right rectus incision. It was noted that considerable mucus was brought up during the operation. The temperature was 101.8, pulse 120, and respirations 30.

The following day his temperature had gone to 103 (101.2 minimum), pulse 130, and respirations were 48 per minute, the white blood cells remaining at 15,000.

Examination of the lungs at this time revealed dullness and diminished excursion on the right side, suppression of breath sounds in the right axilla and right base posteriorly, while over the rest of the lung the breath sounds were bronchial in character. At this time he had a marked cough but no chest pain and was cyanotic in appearance. X-ray examination showed a massive atelectasis (Fig. 4) on the right side.

During the evening the patient was instructed to lie on the uninvolved (left) side. Very shortly after turning the patient on his left side he expectorated a large quantity of sputum which was followed by quite a noticeable decrease in the cyanosis, and he felt definitely better.

On March 9 the maximum temperature was 101.8 degrees, pulse 130, and respirations 42 per minute. There still remained some decrease in excursion on the right side, and the percussion note was dull at the angle of the right scapula. The breath sounds were heard faintly over the involved area, while bronchial breathing was heard over the right apex posteriorly. X-ray examination (Fig. 5) showed definite clearing of the right lung.

The next day the respirations had fallen to 30 per minute, the maximum temperature was 101 degrees, pulse 130, and there was only a slight limitation of expansion on the right, with normal tactile fremitus over the lung except at the extreme right base where it was slightly diminished. The breath sounds were audible over the previously silent area.

On March 11 he was still running an increased respiratory rate of 24 to 32 per minute, but his temperature had dropped to 100 maximum, and pulse to 110. The patient was feeling definitely better and was sitting up in bed. He continued to cough and expectorate a copious amount of mucus. Physical examination now revealed bronchial breathing with coarse crepitant râles over the affected area, with increased tactile fremitus, and X-ray examination showed (Fig. 6) only a slight residue of the massive atelectasis.

During the next few days he continued to improve, and he was discharged on March 24, 17 days after operation.

CASE 3. J. C., Surg. No. 30878. A single white male of 24 entered the hospital on March 8, 1928, for a gastro-enterostomy. Physical examination showed nothing abnormal.

On March 12, 1928, the patient was operated upon under ether anæsthesia, and it was noted that he had a tendency to an accumulation of mucus in his throat. A right rectus incision was made and a resection of the pylorus and first part of the duodenum, with a posterior gastrojejunostomy, (Pólya) was done.

On the first postoperative day the patient's temperature rose to 102 degrees, pulse to 120, respirations to 48 per minute. Examination of the chest revealed some crepitant râles at both bases, more marked on the right. No definite displacement of the mediastinal contents was noted. An X-ray film of the chest (portable) showed an early but definite

Fig. 15.

Fig. 16.

Fig. 17.

Fig. 15. Case 5. There is apparent displacement of the trachea and heart to the right with elevation of the right diaphragm, but no particular clouding of the right lung. The findings suggest a mild degree of postoperative massive atelectasis—not definite due to rotation of patient.

Fig. 16. Case 5. Roentgenogram after postural treatment and cough. There is now definite clearing of the right base and return of the heart and trachea to the midline.

Fig. 17. Case 5. After 3 hours of passive postural drainage, there is definite clearing of the right base, the lower lobe now appearing patchy instead of solid, and the diaphragm is visible. The mediastinal structures have returned to the midline.

massive atelectasis with clouding of the right lung, elevation of the right diaphragm, and displacement of heart and trachea toward the involved side (Fig. 7).

On the second postoperative day the patient had a sharp pain in the right chest, and his temperature fluctuated between 97.2 and 102. The pulse ranged from 90 to 110 and the respirations from 24 to 25. At this time, examination of the chest revealed dullness at the right base, diminished breath sounds and tactile fremitus, and numerous râles could be heard over the whole area. Portable X-rays showed complete de-aeration of the right lower lobe (Fig. 8).

The patient was instructed to lie on his left side and made to cough forcibly. Shortly he commenced bringing up large amounts of tenacious mucus. Following this, the area of dullness over the right lower lobe disappeared and breath sounds became normal. Tactile fremitus became normal, and the patient felt much better. Portable X-rays demonstrated re-aeration of the right lower lobe (Fig. 9) and the following day (Fig. 10) the lung was almost normal in appearance.

The patient was discharged free of symptoms on March 28, 16 days after operation.

CASE 4. M. K. R., Surg. No. 30179. A white female of 13 entered the hospital on November 27, 1927, with an acute attack of appendicitis.

Physical examination showed marked rigidity of the entire abdominal wall, with exquisite tenderness throughout, but more marked on the right. There were a few râles noted in the left chest. Temperature was 102 degrees, white blood cells, 12,300.

Under gas, oxygen, and ether anæsthesia, a low right rectus incision was made and a ruptured appendix was found. A general peritonitis was present. The appendix was removed and ample drainage was provided.

On November 30, it was noted that there were numerous râles over the whole left chest. The temperature varied from 101.6 degrees to 102.6 degrees, pulse 104 to 124, and respirations 35 to 55 per minute.

On December 1, an X-ray film of the chest was taken, which showed a well-established massive atelectasis on the left side (Fig. 11). Portable films of the chest were made daily, and the patient was

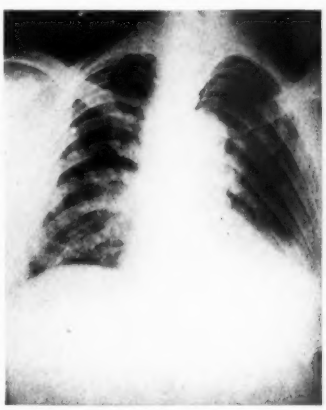

Fig. 18. Case 5. After 18 hours of passive postural drainage the massive collapse has shifted from the right lower lobe to the left lower lobe, and the heart and trachea are displaced to the left side.

considered as a control for the cases treated by postural drainage, as she was too sick to be moved. The massive atelectasis alternately cleared slightly and recurred (Figs. 12 and 13) over a period of 37 days, with three separate and distinct remissions and relapses during this period, and finally disappeared. The patient ran a fever and a high pulse during this period, but the respirations remained elevated only about a week, suggesting that she accommodated herself to the loss of air space rather promptly. The peritonitis was also overcome gradually, and she was discharged on February 14, 1928, after 2½ months in the hospital. A film of the chest just before discharge (Fig. 14) showed no essential variation from normal except that the left diaphragm was limited in mobility.

Case 5. P. L., Surg. No. 30561. A Greek carpenter of 43 years entered the hospital on January 26, 1928, with a diagnosis of duodenal ulcer, confirmed by X-ray examination.

Physical examination was negative except for some spasm and tenderness in the right upper quadrant. White blood cell count showed 12,600.

On January 27, under gas-oxygen and ether anæsthesia, the patient was operated upon and a cholecystectomy performed, no ulcer being demonstrable. Recovery from the operation was uneventful until the third day when the patient presented signs of a pulmonary complication, and a portable roentgenogram of the chest was made which showed a mild atelectatic collapse of the right lung (Fig. 15).

This was confirmed by another film the following day, and on the third day of his postoperative complication, at 6 p.m., careful physical examination revealed the following: Both sides of the chest apparently were symmetrical and moved equally. Percussion showed an area of dullness at the right base posteriorly from about the eighth rib to the base medially, and extending around laterally to the lower part of the axilla. On auscultation no breath sounds or râles could be heard over this area of dullness (Fig. 16).

The patient was then instructed to lie on his left side and to cough whenever he felt like doing so. He remained in this position for 2½ hours, and during this time he coughed considerably and raised a great deal of thick, tenacious sputum. This was not blood streaked.

At 9 p.m. the right chest was re-examined and the percussion note was found to be more resonant than at 6 p.m. On auscultation at this time, the breath sounds were audible over the previously silent area and some coarse bubbling râles could be heard. Another roentgenogram was taken (Fig. 17) which showed partial clearing of the right lower lobe. The patient spent most of the night lying on the uninvolved side, raising mucus at intervals.

On February 4, examination of the chest showed the heart to be in normal position, and the lungs showed no definite evidence of consolidation. The breath sounds were somewhat diminished at *both bases*. A film of the chest showed marked clearing of the right base, but also showed elevation of the *left* diaphragm and partial clouding of the *left base*, with displacement of the heart *toward the left*, indicating an atelectasis on this side; in other words, a transference, due to the patient's position, of the process from right base to left base (Fig. 18). That day the patient passed a tarry stool, there were repeated hæmorrhages the 3 following days, and death ensued on February 8, 12 days after operation. Chest films on the intervening days showed clearing of both bases. Autopsy showed an ulcer on the posterior wall of the duodenum, with a bleeding artery in its base as the cause of death. The lungs were essentially negative except for more intrabronchial secretions than normal, and extensive fibrous adhesions of visceral to parietal pleura on the left. No atelectasis was demonstrable.

The 9 remaining cases in our series were not treated by any particular method and all recovered spontaneously. They will, therefore, be reviewed only very briefly.

Case 6. M. A. S., Surg. No. 23029. A 59 year old woman had a panhysterectomy under ether anæsthesia on January 20, 1925. A low midline rectus incision was used. The onset of the collapse was gradual, the first evidence of a pulmonary complication being a rise of temperature on the seventh postoperative day. The first X-ray film was made on the seventeenth day after operation and revealed a mild collapse and an interlobar pleurisy, both on the right. Re-examination 7 days later still showed some displacement of the heart and mediastinum to the right. She was discharged convalescent the next week, 32 days after operation.

Her maximum temperature was only 100 degrees, pulse 116, and respirations 28 per minute.

Case 7. E. E. G., Surg. No. 23930. A white woman of 26 years, with a history of pleurisy 8 years ago, and bronchopneumonia, had a cholecystectomy performed on May 25, 1925, under ether anæsthesia. A right rectus incision was used. Her first symptom was a spell of coughing and shortness of breath on the second day postoperative, when physical examination demonstrated a diminution of breath sounds over the right lower chest anteriorly and posteriorly, and later a friction rub. X-ray examinations revealed a mild collapse of the right lower lobe and marked clearing the next day, which followed the expectoration of thick mucoid material. A third examination 12 days later showed slight mediastinal displacement persisting. She was discharged 16 days after operation.

Her maximum temperature was 101.2 degrees, pulse 130, and respirations 40 per minute.

Case 8. N. R., Surg. No. 23708. A white boy of 17 had an appendectomy on February 14, 1925, under ether anæsthesia. A muscle splitting incision was used. On the second day after operation the temperature rose suddenly to 102 degrees, pulse to 140, and respirations to 40 per minute. Physical

examination revealed an immobile right chest, cardiac dislocation, dullness over middle and lower lobes on the right, faint and distant breath sounds with bronchial breathing. A portable film of the chest showed almost complete collapse of the right lung, sparing only the apex. Four hours later no breath sounds could be heard through the right chest anywhere except in the infraclavicular region and there only faintly. The white blood cells had risen from 8,000 the day of operation to 30,400 the second day after operation. X-ray films showed beginning clearing the third day after operation, progressing gradually, but there was still a definite residue at last examination on the tenth day. This patient was discharged on the twenty-third postoperative day.

His maximum temperature was 102 degrees, pulse 140, respirations 40 per minute.

CASE 9. B. W., Surg. No. 24969. A man of 64 years had an exploratory laparotomy on October 17, 1925, revealing inoperable lymphoma of the terminal ileum. Ether anæsthesia was used. Cough and considerable sputum were noted on the third day, with a temperature of 102 degrees, pulse 84, and respirations 24. On the sixth day dyspnœa was present, and examination showed dullness all over the right chest, hyperresonance on the left, and cardiac dislocation to the right. Breath sounds were bronchial over the right chest posteriorly, distant anteriorly. X-ray films, October 24, showed massive collapse of the whole right lung, unchanged the following day, with gradual aeration beginning in the lower lobe 6 days later (13 days after operation). The last film 18 days after operation showed the mediastinum in normal position, but the right lung remained cloudy. He was discharged December 3, 17 days after operation.

His maximum temperature was 102 degrees, pulse 98, respirations 24.

CASE 10. M. W., Surg. No. 23387. A white woman of 29 years had a salpingectomy, appendectomy, and ventral suspension of the uterus done on March 11, 1925, under ether anæsthesia. A low, mid-line incision was used. The onset of her collapse came the following day, with cough, cyanosis, and "mucus in the throat." Labored respirations were noted the second day, and examination revealed crepitant râles at both bases with bronchial breathing at the right base. X-ray films showed collapse of the right lower lobe, and the following day showed definite clearing. No further roentgenograms were taken. This patient was discharged on the eighteenth day after operation.

Her maximum temperature was 101 degrees, pulse 140, respirations 30 per minute.

CASE 11. A. P., Surg. No. 25858. A white woman of 50 years had an appendectomy, supracervical panhysterectomy, and a colporrhaphy on March 1, 1926, under ether anæsthesia. The incision was recorded as "supra-pubic." The first symptom was cough on the day after operation, but examination was negative. On the second day the cough was worse, coarse crackling râles were found, and a roentgenogram revealed a mild collapse of the right lung, elevated diaphragm, displaced heart, but only a diffuse clouding on the right, no localized deaeration. She recovered promptly, no further X-ray films being requested, and she was discharged on the eighteenth day after operation.

Her maximum temperature was 101 degrees, pulse 120, and respirations 30 per minute.

CASE 12. A. V. G., Surg. No. 27749. A white woman of 23 years had a Gilliam suspension of the uterus and an appendectomy done on December 9, 1926, under ether anæsthesia, through a mid-line suprapubic incision. The following day she complained of "phlegm" in her throat and her temperature was 102.8 degrees, pulse 128, respirations 24 per minute. The second day after operation a friction rub was found over the entire right chest anteriorly, with numerous fine moist râles and bronchial breathing below the clavicle and in the upper part of the right axilla. She began to expectorate a very thick, purulent but odorless, greenish sputum. X-ray films showed partial collapse of the right lung, with irregular small areas scattered through all three lobes. (X-ray pictures of the lungs before operation had revealed nothing abnormal.) Her condition remained unchanged for 9 days, but no further roentgenograms were taken. She was discharged on the twenty-second day after operation.

Her maximum temperature was 102.8 degrees, pulse 148, respirations 30 per minute.

CASE 13. K. A. C., Surg. No. 25848. A white woman of 22 years had an appendectomy done on March 1, 1926, under ether anæsthesia, through a right rectus incision. The first symptom of a postoperative pulmonary complication came 48 hours after operation, with a sore throat, purulent sputum, and pain in the right lower chest. Physical examination revealed moderate dullness on percussion over the right base posteriorly, bronchial breathing over this area, and a friction rub, but no displacement of the heart was noted. A portable film of the chest showed clouding of the right base with some displacement of the heart and trachea to the right. In addition, there was a small area of increased density in the left upper lobe laterally. No further X-ray examinations were called for as she improved rapidly, coughing up considerable purulent sputum meanwhile. She was discharged 13 days after operation.

Her maximum temperature was 101 degrees, pulse 120, and respirations 40 per minute.

CASE 14. D. E. M., Surg. No. 28408. A white male of 27 years was operated upon for gastric perforation on March 14, 1927, under ether anæsthesia, a right rectus incision being used. The perforation was found and closed, no gastro-enterostomy being done. Early on the second postoperative day the patient suddenly became dyspnœic, was very cyanotic, and his breathing was labored. He was lying on his right side. Physical examination at this time showed marked limitation of respiratory move-

ment on the right, with increased respiratory excursions on the left. Tactile fremitus was present on both sides. The percussion note over the right lower lobe anteriorly was dull, and there was bronchial breathing over this area. A roentgenogram showed the lower half of the right chest to be opaque, obscuring the right diaphragm, and the heart and trachea were displaced to the side side. A reexamination the following day showed more displacement of the mediastinal contents, but the clouded area remained unchanged. The signs and symptoms lasted for 5 days, slowly decreasing in intensity, no further X-ray films being made. He was discharged April 1, 1927, 16 days after operation.

The maximum temperature was 102 degrees, pulse 140, and respirations 42.

The cases reported above have afforded us an opportunity to obtain a few facts which are significant, i.e., Cases 3 and 5 have shown quite definitely that the first step in the production of the disease is the occlusion of the air passages, followed by the absorption of the air, and then the next step is the displacement of the mediastinal contents towards the affected side, and an elevation of the diaphragm. Then, with the absorption of more air from the alveoli and conditions for further displacement of the diaphragm and mediastinal contents becoming more difficult, there is a pouring out of secretions from the cells lining the bronchi, and an exudation of serum through the vessel walls into the bronchi where a tendency toward a negative pressure exists. The obstructing material which is essential to the production of the disease is the thick, tenacious mucus which does not form a mucus plug, but rather more probably a diaphragm of mucus over the lumen of the air passages. Clearing of the consolidated area occurs first when the condition is abating, and this is followed by a return of the mediastinal contents to their normal position.

Case 4 has shown that we may have a recurring massive atelectasis. This patient had three definite spontaneous remissions and relapses, but the disease (untreated) lasted 37 days.

Case 5 demonstrates that massive collapse may be transferred from one lobe to another lobe, the deciding factor being gravity. Case 5 also proves that massive atelectasis may occur in one lung even though the opposite lung is restricted by extensive pleural adhesions.

We wish to call attention to the frequency of this disease, the methods of prevention, the postural method of treatment, the infrequency of clinical diagnosis (2 out of 14), and the ease of recognition if roentgen examination is made routinely in all cases with postoperative pulmonary complications.

CONCLUSIONS

1. Obstruction to the air passages is absolutely essential for the production of the disease. Thick tenacious mucus is the obstructing material.

2. Usually many secondary factors are involved in the production of the disease, i.e., lowered vital capacity, raised cough re lex, lack of frequent postural change after and during operation, and limitation of thoracic and abdominal mobility due to the operation.

3. The disease is not a reflex nervous phenomenon.

4. The condition does not occur contralateral to the side operated upon unless the patient lies on his side during the operation, as in renal operations.

5. The Sante maneuver is very efficacious in treating the disease.

6. Hyperventilation of the lungs with carbon dioxide and oxygen should be employed at the end of operation and during the first 48 hours postoperative.

7. Frequent changes of posture should be made during the first few days after operation, and sedatives should be curtailed.

8. The mortality from the disease is very low.

Since the original presentation of this material before a clinic of the American College of Surgeons in Boston, October 10, 1928, several articles on the subject have appeared. Of prime importance are the ones by Bowen (Am. J. Roentgenol. & Radium Ther., 1929), and by Lee, Tucker, Ravdin, and Pendergrass (Arch. Surg., Jan., 1929). The latter gives a complete account of the experimental production of this disease in dogs, and proves beyond a doubt that obstruction of the air passages is the primary factor in the causation of postoperative massive atelectatic collapse, and that the important contributory factor is the abolition of the cough reflex.

BIBLIOGRAPHY

1. Bergamini, H., and Shepard, L. A. Bilateral atelectasis (massive collapse) of lung. Ann. Surg., 1927, lxxxvi, 35.

2. Bradford, J. R. Massive collapse of the lung as a result of gun shot wounds; with special reference to wounds of the chest. Quart. J. Med., 1918, xii, 127.
3. Bradford, Sir J. R. Oxford Loose Lead Medicine, 1920, ii, 127.
4. Briscoe, J. C. Quart. J. Med., 1920, xiii, 293.
5. Crymble, P. T. Brit. J. Surg., 1918, v, 363.
6. Cutler, E. C., and Hunt, Alice M. Arch. Int. Med., 1922, xxix, 444.
7. Elliott, T. R., and Dingley, L. A. Lancet, 1914, i, 1205.
8. Elwyn, H., and Girsdansky, J. J. Am. M. Ass., 1922, lxxix, 718.
9. Endes, M. F. Postoperative massive atelectasis; report of cases. Boston M. & S. J., 1926, cxcv, 258.
10. Hahn, L. J. Massive collapse of the lung following nephrectomy. Internat. J. Med. & Surg., 1924, xxxvii, 232.
11. Harrington, S. W. Relief of postoperative massive collapse of the lung by bronchoscopic aspiration. Ann. Surg., 1927, lxxxv, 152.
12. Harris, H. A. Atelectasis of lung (Case 7). Canadian M. Ass. J., 1925, xv, 808.
13. Hearn, W. P., and Clerf, L. H. Postoperative massive collapse of the lung. Ann. Surg., lxxxv, 54.
14. Hirschboech, F. J. Postoperative massive collapse of the lungs. Am. J. M. Sc., 1922, clxiv, 268.
15. Holmes, George W. Am. J. Roentgenol. & Rad. Therap., 1924, xi, 509.
16. Hunt, E. L. Massive atelectasis of lung as a surgical complication (Case 1). Boston M. & S. J., 1926, cxciv, 58.
17. Jackson, Chevalier, and Lee, Walter E. Ann. Surg., 1925, lxxxii, 364.
18. Pasteur, W. Am. J. M. Sc., 1890, c, 242.
19. Idem. Royal College of Physicians. Bradshaw Lecture, 1908.
20. Idem. Brit. J. Surg., 1914, i, 587.
21. Powers, J. H. Vital capacity. Arch. Surg., 1928, xvii, 304–323.
22. Regan, J. C. Lancet, 1924, ii, 1222.
23. Ritvo, Max. Am. J. Roentgenol. & Rad. Therap., 1924, xi, 327.
24. Sante, L. R. Massive collapse of the lung. Radiol., 1927, viii, 1.
25. Idem. Massive (atelectatic) collapse of lung; with especial reference to treatment. J. Am. M. Ass., 1927, lxxxviii, 1539.
26. Idem. Massive (atelectatic) collapse of the lung. Ann. Surg., 1928, lxxxviii, 161.
27. Santee, H. E. Bilateral massive collapse of the lung. Ann. Surg., 1927, lxxxv, 608.
28. Scott, W. J. M. Arch. Surg., 1925, x, 73.
29. Scott, W. J. M., and Cutler, E. C. Postoperative massive atelectasis. J. Am. M. Ass., 1928, xc, 1759.
30. Scrimger, F. A. C. Surg., Gynec. & Obst., 1921, xxxii, 486.
31. Tidy, H. L. Acute lobar collapse of the lung. Lancet, i, 1245.

TUMORS OF THE CAROTID BODY

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THE carotid body was first described as the "Ganglion minutum" by von Haller in 1743. This knowledge soon was forgotten, but Neubauer in 1783 once more brought it to light. Shortly thereafter, 1797, Andersch gave a good description and named the body at the bifurcation of the common carotid artery the "Ganglion intercaroticum." Interest again waned until 1862 when Luschka published his splendid report containing detailed microscopic studies of this peculiar structure. Since that time it has been extensively studied by many men and its various characteristics are gradually becoming clear, although many points are still a matter of dispute. A patient with carotid tumor was recently admitted to our service, and this gave us the opportunity of studying this condition.

The patient (Mr. E. T. M.), 39 years of age, was referred to Dr. Bevan by a physician who for some time had been treating him for syphilis. While he was under specific treatment he developed a nodule in the left side of the neck just below the angle of the jaw; the nodule pulsated. It was at first small and just palpable. It increased gradually in size until it was about as large as an English walnut. There had been very few symptoms in connection with the nodule, possibly some tingling or twinges, hardly severe enough to classify as pain. The medical man who had him under treatment for syphilis for a period of 3 years, from 1923 to 1926, thought at first that it might be specific, but it did not disappear under treatment and he then concluded that it might be a tuberculous gland, and referred him for operation and treatment.

We also thought that under the circumstances the most probable diagnosis was a tuberculous gland, although we felt that the diagnosis was uncertain. We advised an exploratory operation, which was made last October under local anæsthesia. A very thorough physical examination was made before the operation. The mass which was the size of a walnut gave one the impression of pulsation. It seemed, however, that when the pulsation was examined carefully it probably was a transmitted pulsation from the carotid and not the expansile pulsation of an aneurism, although the possibility of aneurism had to be considered. There was another feature about the case that impressed us; that was that the mass was very freely movable from side to side but not from above downward. The operation was performed under local anæsthesia. The operative field was infiltrated with novocain and adrenalin solution. An incision on the inner side of the sternocleidomastoid, dividing the skin and superficial fascia, the platysma, and the deep fascia surrounding the entire neck, was made. Making a very careful and complete dissection we exposed the common carotid for the distance of about 2 inches and the internal carotid and the external carotid to a point above the tumor. At the crotch of the common carotid, a tumor about the size of an English walnut, of a brownish, purplish red color was found. The mass looked as though it was exceedingly vascular and made up largely of loops of blood vessels. From its location and the character of the tissue it was quite evident that we had to deal with a tumor of the carotid body. The tumor involved the wall of all three carotids, the common, the external, and the internal. The sketch of the neck was made by Mr. Shephard at the time of the operation (Fig. 1). The tissue between these great vessels and the tumor was infiltrated with novocain solution and an attempt made to dissect the tumor from the blood vessel walls, but it was found that it would be impossible to make a complete removal of the tumor without removing the common carotid and the internal and external carotids to which it was attached.

One of us, Dr. Bevan, has done a great many ligations of the three carotid vessels, possibly one hundred or more. The great majority of these have been ligations of the external carotid as a preliminary step in the amputation of the tongue, in removing extensive carcinomata from the mouth, and in resecting the upper jaw, also extensive carcinomata of the face and the side of the neck. Probably 80 per cent of these carotid ligations have been operations of this kind in which the ligation of the external carotid was done for the purpose of controlling hæmorrhage.

The common and internal carotids were also ligated in possibly twenty-five cases. About half of these have been cases of pulsating exophthalmos due to skull fractures and injury of the internal carotid in the cavernous sinus. The other half of the ligations have been in cases of extensive neck dissections in malignant disease, and a few of them in very extensive vascular neoplasms of children in which it was necessary to ligate both the common carotid and internal jugular, in which the vascular

neoplasm was supplied by both the internal and the external carotid.

These facts are cited because this experience has made us very familiar with the enormous mortality that results from ligation of the common carotid and internal carotid from cutting off the blood supply of that side of the brain, with a resulting necrosis of the brain tissue and death of the patient. There is, as is well known, an enormous difference in the risk of ligating an external carotid from that of the internal carotid or common carotid. We have had practically no mortalities in our very large series of external carotids; on the other hand, we have had a number of fatal and serious accidents in ligating the common and the internal, the ligation of the internal being the most serious of the three. Statistics covering a large number of cases show that in the neighborhood of 30 per cent of ligations of the common and internal carotids are followed by death of the patient.

As we stood in the operating room with this patient on the table, with the tumor and the carotid vessels exposed, these facts flashed through our minds. The patient was under local anæsthesia, so without any hardship to him one of the assistants went down to the library and brought up a volume of Keen's *Surgery* containing an article on tumors of the carotid body which one of the assistants read to the operating group.

Although it was a great temptation, with the complete dissection which had been made, to remove this tumor, the "Golden Rule" was applied. Ask yourself what you would want done with your knowledge as a surgeon under these circumstances if you were the patient, and you will recognize the fact at once that you would not want your carotid vessels ligated and take the chance of 30 per cent mortality from the operation with the added chance of hemiplegia following the operation even though you survived it. We felt very positive in our position when we realized that the majority of these tumors are not malignant and the patients live for 20 to 30 years or longer with tumors of the carotid body without ever becoming malignant or without their producing symptoms of moment. We, therefore, made the operation purely exploratory, closed the incision, and decided to see what could be done by radiation in the way of reducing the size of the tumor or possibly bringing about a cure.[1]

This is the first carotid tumor that we have operated upon. Very few surgeons have operated upon more than one, a few have operated upon two cases, and no one has reported more than three. In other words, no one has had a large enough experience to dogmatize on the subject, at least from the standpoint of his own limited experience with these cases. We felt, therefore, that it would be wise to review the entire literature of the subject for the purpose of determining the best method of handling these cases.

ANATOMY

The carotid body is of variable size, from 5 to 7 millimeters long, 2.5 to 4 millimeters wide, and 1 to 1.5 millimeters thick. Its location is not absolutely constant, at times being in the middle of the bifurcation of the common carotid artery, at others directly behind it, while sometimes it lies on the medial-posterior surface of the internal carotid just at the bifurcation. It has a definite fibrous capsule and is connected to the wall of the carotid, either internal or external, by a fibro-adipose pedicle about 2 or 3 millimeters long, called the ligament of Mayer. It is through this pedicle that the nutrient vessels run. The color varies from gray-red to purple-red, depending upon the amount of blood present. It is usually homogenous with a smooth surface but sometimes it is broken up by septa into many loosely connected parts. The arteries to the gland by most authors are stated to come from the common or internal carotids. This is doubtful, however, in view of the recent splendid work of Smith who shows that in all animal embryos studied, except the pig and including the human, the artery is derived from the external

[1]November 8, 1929. Thirteen months have now elapsed since the operation. The patient was observed today and states that he has had absolutely no symptoms referable to tumor. The mass has almost entirely disappeared. The carotid pulsation can be felt much more distinctly on this side than on the other because at the operation the deep cervical fascia was divided for the extent of 4 or 5 inches parallel with the incision. This has permitted the common carotid and its point of division into the internal and external to come much nearer to the surface. To date the patient has had fourteen X-ray treatments. The result of these treatments must be accepted as having produced practically a cure, and it seems to point to the conclusion that treatment with the X-ray, with its very slight danger, is much to be preferred to the operative treatment of these tumors which carries with it the enormous mortality of more than 30 per cent.

carotid and that the branch comes off before the origins of the occipital and ascending pharyngeal. The veins are variable but usually come off the upper pole of the gland and empty into the superior laryngeal, pharyngeal, or lingual. The nerves supplying the carotid body, and with which it is richly endowed, are derived from the pharyngeal branch of the ninth nerve and the superior cervical sympathetic ganglion and the sympathetic trunk. Some authors claim that it has branches from the vagus and hypoglossal as well but this is not proved.

HISTOLOGY

From the inner surface of the fibrous connective tissue capsule, fibrous septa enter and divide the gland into lobules which in turn are divided into lobuli by smaller trabeculæ of the same connective tissue network. This fibrous network is rich in nuclei and many medullated and non-medullated nerve fibers as well as ganglion cells are present. The lobuli are made up of clusters of cells of different types. The majority are large polyhedral cells rich in protoplasm which is finely granular and poor-staining. The individual cell walls are sometimes difficult to distinguish. The nuclei are large, round or oval, somewhat eccentrically placed, and deep-staining. Some authors state that scattered throughout the cellular elements are cells having an affinity for the salts of chromium, but this is disputed. There are also scattered sympathetic ganglion cells, plasma cells, and eosinophilic cells present. There is no regularity of cell arrangement.

There is an exceedingly rich capillary network which in places enlarges to form large sinuses. The lining layer of endothelial cells is thin and the lobuli cells are not separated from them by stroma. Many times these capillaries are flexed upon themselves and become hunched. This gives the appearance of a glomerulus and has given rise to the name "glomo-caroticus." (Fig. 1.)

It must be remembered that the histological picture of the carotid gland undergoes a change with the age of the individual. In childhood and youth the gland is small—about the size of a millet-seed—and the cellular elements predominate. As the individual grows older, the gland also grows larger, to about the size of a bean, and the capillaries and stroma increase at the expense of the cellular elements.

EMBRYOLOGY

There is a marked difference of opinion concerning the origin of the carotid body. The different theories may be grouped under three headings: (1) epithelial; (2) vascular; (3) nervous. The advocates of the first of these theories believed that the carotid body was derived from the pharyngeal epithelium. There is at present no evidence supporting this theory. The second or vascular origin is upheld by Arnold, Waldeyer, Schaper, and others. They believe that the body is derived from the blood vessel wall, either endothelial or perithelial. Waldeyer introduced the term perithelial body to differentiate it from endothelial. The nervous origin is upheld by Stilling, Kohn, and others. These believe that the development is from sympathetic ganglion cells of the intercarotid plexus. In a detailed study of the origin and development of the carotid body, Smith concludes that it arises from a complex of materials which become associated during the developmental history of the third mesodermal arch. A portion of the mesenchyme of the third arch should be regarded as the anlage of the mesodermal constituents of the body and not as a localized area of the wall of the carotis interna. The nervous elements are derived from a pharyngeal branch of the glossopharyngeal nerve as well as from the cervical sympathetic and sometimes from vagus. This phase of the subject needs much further study.

PHYSIOLOGY

Relatively little work has been done in this field, and that which has been done is almost barren of results. Moulon, Gomez, Scaffidi, Vassale, Lanzillatta, and Frugoni all did work the results of which in some cases conflict. Moulon, in 1904, prepared an extract from the carotid gland of horses, injected it into rabbits, and noted a rise in blood pressure and acceleration of the pulse. He concluded that it acted like adrenalin. Gomez, in 1907, injected a glycerine extract of the body into cats and found a fall in blood pressure.

Scaffidi destroyed the carotid bodies of calves and observed no ill results. Vassale destroyed the carotid bodies of cats on both sides with cautery and immediately thereafter noted a glycosuria which persisted for about 4 days. The animals never again developed a glycosuria but gradually became cachectic, lost their fur, and died about 6 months after operation. Lanzillatta observed a transient glycosuria from destruction of the sympathetic trunk or superior sympathetic ganglion. Frugoni has shown, by perfusion experiments in rabbits using a calf's carotid body perfusate, that this perfusate has a vasodilatory effect. If such an extract is injected intravenously there is a short initial rise, then a marked fall, in blood pressure with a gradual rise to normal. Fischer extirpated both carotid bodies from young cats. The bones of these animals developed a condition resembling osteomalacia and the parathyroids hypertrophied. There is no definite evidence that the carotid body has any endocrine significance. The results of extirpation experiments are nil, because it is impossible to remove the carotid body without severe trauma to the cervical sympathetic which, of course, clouds any results that may be obtained. Injections and perfusions of extracts by Frugoni as above, gave results that could be explained on other bases. Its marked vascularity suggests an active metabolism.

PATHOLOGY

The only congenital anomalies noted are those of differences in shape or location. It may be a single body divided into many nodules by the capsular septa or there may be several separate lobes only loosely connected by a fascia. Likewise it may be in the fork of the carotid or on the posterior surface of the internal or external carotid artery.

Paunz has noted hæmorrhages in the newborn, especially in premature babies and associated with hæmorrhages in other neck structures. He has also noted them following thyroidectomy and in purpura, especially purpura hæmorrhagica.

Gomez reports a few cases of sclerosis by which he means an increase in the connective tissue stroma. He states that this occurs in old age in individuals with syphilis of the carotid artery and that the sclerosis is directly proportional to the amount of sclerosis of the intima of the vessel. This vessel sclerosis also affects the interlobular vessels in the carotid body, the lobuli cells atrophy, the stroma increases, and may even undergo hyaline degeneration. Schaper claims that these changes are due to age entirely (Paunz reports these changes present in individuals dying of cirrhosis of liver). In the 50 postmortem cases examined by Gomez, he found one case of cloudy swelling in a patient dying of lobar pneumonia. In another case, dead of chronic nephritis, he found a lymphoid infiltration affecting only one lobule. Paunz also found round cell infiltration in hydrophobia. Amyloid degeneration of the small vessels has been noticed.

Deitrich and Siegmund state that suppuration, abscess formation, and phlegmon have occurred.

With these exceptions the carotid body seems to be peculiarly unaffected by general disease processes. It is, however, not uncommonly the site of tumor formation. These tumors have been called various names: endothelioma, perithelioma, perithelial hæmangioma, adenoma, paraganglioma, neuroblastoma, fibro-angioma, hamartoma, phaochromozytoma, and others. Perithelioma suggested by Paltauf and based on the presumed origin of the carotid body seems to be the choice of most pathologists. These tumors are essentially a hyperplasia of the cells of the normal carotid body. In almost all cases they produce on a larger scale the normal histological picture of the cell groups of large, pale-staining cells containing well defined oval nuclei and with more or less indefinite limiting membranes, the groups separated by fibrous trabeculæ and having a rich capillary network as well as many ganglion cells. Occasionally giant cells are present. There may be round cell and plasma cell infiltration and there may also be areas of degeneration with hæmorrhage. There is no regular arrangement of the tumor cells. Some cases are definitely malignant, and in these tumor cells may be found infiltrating the vessel walls and invading the

capsule. Kopfstein, Keen, and Kretschmer report cases with regional lymph gland involvement. Gilford and Davis report one case with metastases in the liver, but it is questionable if this case is one of carotid body tumor as is also true of the patient of Moenckeberg who died of papillary adenosarcoma of the ovary 4 years after removal of a carotid body tumor. These are the only two cases reported of metastases. There are 24 cases (17.8 per cent) of malignancies reported. There are 12 cases (8.9 per cent) of definite recurrences in the literature. Some of the cases reported as malignant may not have been so because of the disagreement as to the type of tumor. This is illustrated by two cases of Burge and one of Fowler. Unless tumor cells can be demonstrated invading the tumor capsule or vessel walls, with or without regional lymph gland involvement, it is very difficult if not impossible to make a diagnosis of malignancy in these cases. The accompanying photomicrographs are made from specimens of the tumor removed by Dr. Carl B. Davis in 1920, and reported by Dr. E. F. Traut in 1927. They show the characteristic features of the tumor (Figs. 5 and 6).

DIAGNOSIS

A correct pre-operative diagnosis of these tumors is seldom made. Only fourteen such diagnoses are on record. This is mainly because of the rarity of the tumor. No one man has ever had more than three cases come under his observation. In all cases there is a visible tumor mass usually in the upper anterior cervical triangle with the posterior margin under sternocleidomastoid muscle. This tumor mass is of variable size, some being as small as a hazelnut, others as large as a goose egg. Usually they are of hen's egg size when the patient first consults a physician. The mass practically always pulsates but the pulsation is not that full expansion and contraction of an aneurism but rather a sort of transmitted pulsation. In most cases firm pressure on the mass will considerably diminish its size, since this forces the blood out of the highly vascular mass. There may be a thrill and a bruit. The skin of the neck is unchanged and it is freely movable over the tumor mass. The mass itself is movable laterally but not up and down and is almost always unattached to surrounding structures. The surface is usually smooth but in several cases it has been nodular. Practically never do patients with carotid tumors complain of pain, although there may be occasional tingling sensations in the neck. The mass is not tender to palpation.

The associated symptoms that may be present are almost all pressure effects. The vagus, cervical sympathetic, and recurrent laryngeal are the structures most commonly thus affected. Knighton reports a case in which the patient had fainting spells due to vagus pressure and Boot reports one with Stokes-Adams syndrome. Hoarseness and cough are relatively common. Dysphagia, dyspnœa, tinnitus aurium, headache, dilatation of the pupil have been noted.

The tumor is practically always unilateral. It occurs at any age; the youngest case on record is 7 years, and the oldest is 73. It is most frequent between the ages of 30 to 40 and practically 70 per cent of the cases occur between 30 and 60 years. The average age of 126 patients was 41.7 years.

In 131 cases that have recorded the sex of the patients, 64 cases were males and 67 were females.

TABLE I.—ANALYSIS OF CASES

AGE INCIDENCE

Years	Cases	Per cent
1 to 10	1	
10 to 20	5	19.2
20 to 30	18	
30 to 40	36	
40 to 50	27	69.6
50 to 60	24	
60 to 70	13	11.2
70 to 80	1	

SEX INCIDENCE

Males	64
Females	67

Average age of patients, 41.7 years.
Average duration of tumor before consulting physician, 7 years, 2 months.
Total cases reported, 143.
Number of postmortem cases, 9.
Total cases reporting results of operations, 125.
Total deaths, malignancies included, 44 or 35.2 per cent.
Total deaths, postoperative, 27 or 21.6 per cent.
Total malignancies and recurrences, 24 or 17.8 per cent.
Total recurrences, 12 or 8.9 per cent.
Total cases reported recovering, 90.

Total cases with various postoperative disabilities, 39 or 43.3 per cent.
Total cases with common or all carotids ligated, 62.
Total number of deaths in these cases, 21 or 33.8 per cent.
Correctly diagnosed pre-operatively, 14.

From an analysis of this entire group, it is clearly shown that an overwhelming majority of tumors of the carotid body are benign, certainly more than 80 per cent; possibly not more than 15 per cent are malignant. Among malignant cases reported there are certainly a number which careful analysis would exclude from this group as they are probably lymphosarcomata and other malignant tumors which happen to involve the region of the carotid body, but certainly a number of them did not originate in the carotid gland but in the surrounding tissues. As shown in Table I in more than 30 per cent of the patients operated upon in which it was necessary to ligate the carotid artery the result has been fatal, and in a certain percentage ligations have been followed by aphasia and hemiplegia, usually permanent in character.

SUMMARY AND CONCLUSIONS

With this evidence we wish to present the conclusion that in the future neoplasms of the carotid body should not be removed when it is necessary to ligate the carotid arteries in order to complete the operation. If the common carotid and the internal carotid can be saved by careful dissection, done best under local anæsthesia, the removal of a benign tumor of the carotid gland would be justified. If the surgeon had definite and satisfactory evidence that the tumor was malignant, the huge 30 per cent mortality involved in the ligation of the carotid arteries might be accepted in order to save the patient from death and from malignant disease. It is quite evident, however, that a malignant growth of the carotid gland involving these three arteries offers very little prospect of a permanent cure even by the most extensive operation.

We believe that these conclusions should be generally accepted and that they should control the actions of the surgeons who in the future are confronted, as they usually are, unexpectedly with this problem. It is true, of course, that in very few of these cases up to the present time has diagnosis been made before operation; in practically none of them has a definite diagnosis of tumor of the carotid been made. With the amount of evidence which we now have and which has been accumulating in the last 20 years, the clinical diagnosis will be made more frequently in the future from three points: the location of the tumor at the crotch of the carotid; the sense of pulsation in the tumor; and the fact that the tumor is very movable from side to side but is not movable at all from above downward, and the added fact that the tumor has existed for many months or even years before the patient seeks surgical relief.

Case 1. Operation by Reigner (67), 1880.

Female, aged 32 years, had a tumor 4½ years. All carotids, internal jugular and pharyngeal arteries were ligated. Vagus and sympathetic nerves were injured. Clinical diagnosis was lymphoma but growth was proved to be malignant on pathological examination. Patient died of bronchopneumonia 3 days after operation.

Case 2. Operation by Maydl (73), 1886.

Male, aged 28 years, had a tumor 6 months. All carotids, pharyngeal and superior thyroid arteries were ligated. No clinical diagnosis was made. Pathological diagnosis was perithelioma. Postoperative aphasia and hemiplegia were unchanged 4 years later.

Case 3. Operation by Dittel (73), 1886.

Male, aged 32 years, had a tumor for several months. All carotids and internal jugular arteries were ligated. No clinical diagnosis was made. Pathological diagnosis was perithelioma. Patient died of secondary hæmorrhage.

Case 4. Operation by Gersung (73), 1886.

All carotids and pharyngeal arteries ligated. Clinical diagnosis was tuberculous glands of the neck. Pathological diagnosis was perithelioma. Patient recovered but suffered from paralysis of left vocal cord. This condition remained unchanged 4 years later.

Case 5. Operation by Albert (73), 1889.

Male, aged 35 years, had a tumor for 5 years. External carotid was ligated. No clinical diagnosis was made; pathological diagnosis, perithelioma. There was a recurrence one year later. Recurrent tumor was removed but patient was lost track of.

Case 6. Kauffman and Ruppanner (49), 1891.

Female, aged 25 years, had a tumor for 7 years. Common and internal carotids and internal jugular were ligated. Vagus and hypoglossus nerves were injured. No clinical diagnosis was made but pathological diagnosis was carotid tumor. Hoarseness and contraction of pupil followed operation and patient died of pneumonia 2 days after operation.

Case 7. Kretschmer (56), 1892.

Male, aged 48 years, had a tumor 1 year. Superior thyroid, lingual arteries, and external jugular were

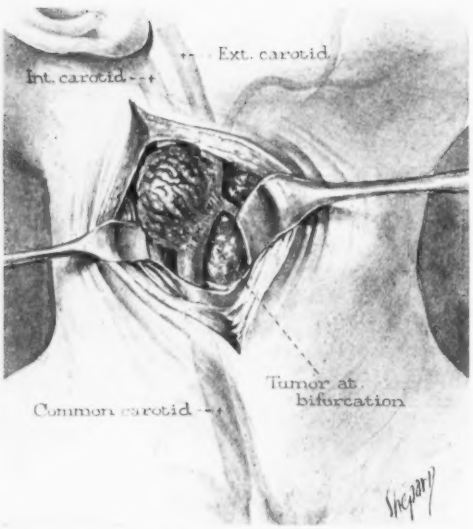

Fig. 1. Tumor at bifurcation of common carotid artery. (Semi schematic.)

ligated. Clinical diagnosis was branchial carcinoma but pathologically the growth was found to be a carotid tumor. There was postoperative paralysis of the left vocal cord. Recurrence took place in 4 months. Attempt was made to remove the recurrence but it was impossible. Patient was alive 14 months later.

CASE 8. Maydl (55), 1893.

Female, aged 46 years, had a tumor for 16 years. All carotids, pharyngeal arteries, and internal jugular were ligated. The sympathetic nerve was injured. Clinical diagnosis was carotid tumor. Pathological diagnosis was endothelioma. Patient recovered but mydriasis was marked.

CASE 9. Maydl (55), 1895.

Male, aged 34 years, had a tumor 4 years. All carotids, pharyngeal, superior thyroid, lingual, and external maxillary arteries were ligated. The vagus, facial, hypoglossus, and sympathetic nerves were injured. The clinical diagnosis was carotid tumor and the pathological diagnosis was malignant tumor of endothelial type. Patient recovered from operation but suffered from aphonia and paralysis of the tongue and left vocal cord.

CASE 10. Malinowsky (66), 1895.

Female, aged 30 years, had suffered from a tumor for 10 years. All carotids and internal jugular were ligated. Eight centimeters of vagus was resected. The clinical diagnosis was lymphosarcoma; pathological diagnosis, perithelioma. Patient recovered.

CASE 11. Middleton and Bierring (69), 1895.

Male, aged 43 years, had a tumor for 6 months. The external carotid was ligated. No clinical diagnosis was made but the pathological diagnosis was carotid tumor. Patient recovered from operation but suffered from paralysis of the palate. Recurrence took place 2 months later and a tumor also appeared on the other side of the neck. Death occurred 3 months after operation.

CASE 12. Moenckeberg (71), 1895.

Male, aged 52 years, had a tumor for 2 years. All carotids and the internal jugular were ligated. The vagus and hypoglossus nerves were injured. No clinical diagnosis was made but the pathological diagnosis was carotid tumor. After operation patient suffered from hemiplegia and aphasia. He died from pneumonia.

CASE 13. Moenckeberg (71), 1895.

Female, aged 30 years, had a tumor for a year and a half. All the carotids were ligated. No clinical diagnosis was made but the pathological diagnosis was carotid tumor. Patient recovered from operation but had a right facial paralysis. Death occurred 4 years later from papillary adenosarcoma of ovary.

CASE 14. Moenckeberg (71), 1898.

Female, aged 50 years, had a tumor for 3 years. All carotids and the superior thyroid were ligated. No clinical diagnosis was made but the pathological diagnosis was carotid tumor. Patient recovered.

CASE 15. Heinleth (45), 1900.

Male, aged 60 years, had a tumor for 37 years. No arteries or nerves were injured at operation. No clinical diagnosis was made but the pathological diagnosis was perithelioma. Patient recovered and there was no recurrence after 4 years.

CASE 16. Dobromidoff (26), 1900.

Male, aged 41 years, had a tumor for 17 years. All carotids and the internal jugular were ligated. The vagus was injured. The clinical diagnosis was tumor of the thyroid; pathological diagnosis, carotid gland tumor. Death occurred one day after operation.

CASE 17. Siniouschine (89), 1901.

Female, aged 41 years, had a tumor for 8 years. All carotids and internal jugular were ligated. The facial nerve was injured. Clinical diagnosis was fibrolipoma but pathological examination showed the tumor to be either an endothelioma or a perithelioma. Patient recovered.

CASE 18. Scudder (85), 1902.

Female, aged 32 years, had a tumor 9 years. All carotids were ligated. No clinical diagnosis was made but the pathological diagnosis was perithelioma. Patient recovered and had no recurrence in 4 years.

CASE 19. Kauffman and Ruppanner (49), 1902.

Female, aged 21 years, had a tumor 2½ years. Clinical diagnosis was lymphoma or goiter; reported as carotid tumor. Patient recovered and had no recurrence in 4 years.

CASE 20. Reclus and Chevassu (76), 1903.

Female, aged 49 years, had a tumor for 5 years. No ligations but sympathetic and branch of facial nerve injured. Clinical diagnosis, carotid tumor

and pathologically perithelioma. Recovery. Lower branches of facial paralyzed.

CASE 21. Macphater (64), 1903.

Female, aged 34 years, had a tumor 6 years. All carotids and the internal jugular were ligated. No clinical diagnosis was made and pathologically the type of tumor was undetermined. Recovery; no complications.

CASE 22. Cuneo and Dainville (23), 1903.

Female, aged 23 years, had a tumor 3½ years. No ligations were made but the facial nerve was injured. No clinical diagnosis was made. Pathological examination showed carotid tumor. Recovery.

CASE 23. Funke and Hearn (35), 1903.

Male, aged 48 years, had a tumor 6 years. All carotids and the internal jugular were ligated. The clinical diagnosis was fibroma or lipoma but the pathological diagnosis was perithelioma. Patient recovered from operation but died 2 months later of cerebral anæmia.

CASE 24. Gilford and Davis (36), 1904.

Male, aged 52 years, had a tumor of 3 months' standing. Internal jugular was ligated, and the vagus and descendens hypoglossi were injured. No clinical diagnosis was made; pathologically, tumor was an endothelioma. There was a recurrence in 6 weeks and death occurred in 5 months.

CASE 25. Gilford and Davis (36), 1904.

Male, aged 62 years, had a tumor 6 months. All carotids and the internal jugular were ligated. The vagus and descendens hypoglossi were injured. No clinical diagnosis was made but the pathological diagnosis was carotid tumor. Hemiplegia followed operation and death occurred from pneumonia the seventh day after operation.

CASE 26. Gilford and Davis (36), 1904.

Male, aged 74 years, had a tumor 1 year. He died in 18 months of exhaustion. Metastases were found in the liver.

CASE 27. Keen (50), 1905.

Male, aged 56 years, had a tumor 18 years. All carotids and the internal jugular were ligated. No nerves were injured. Clinical diagnosis was lipoma. Pathological diagnosis was perithelioma. Death occurred 2 days after operation from œdema of the lungs.

CASE 28. Dobromidoff (26), 1906.

Female, aged 25 years, had a tumor 7 years. All carotids and many veins were ligated. No clinical diagnosis was made but the pathological diagnosis was carotid tumor. Patient recovered but the pupil remained dilated and conjunctiva red.

CASE 29. DaCosta (24), 1906.

Male, aged 52 years, had a tumor 20 years. All carotids and the internal jugular were ligated. The superior laryngeal nerve was injured. No clinical diagnosis was made but pathological diagnosis was carotid tumor. Recovery. Patient suffered from hoarseness and hemiplegia 8 days after operation.

CASE 30. Lilienthal (60), 1906.

Female, aged 38 years, had a tumor 10 months. First operation was only a biopsy; later one-half of tumor was removed. No clinical diagnosis was made but the pathological diagnosis was perithelial hæmangioma. The growth recurred but there were no metastases. Death took place 9 months later from repeated hæmorrhage and cachexia.

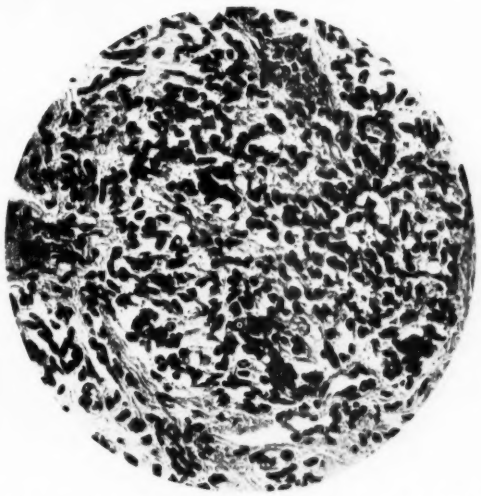

Fig. 2. Normal carotid body. High power. By H. Klose (52).

CASE 31. Graham and Crile (38), 1906.

Male, aged 27 years, had tumor 10 years. All carotids were ligated. The vagus, sympathetic, and recurrent laryngeal nerves were injured. No clinical diagnosis was made but the pathological diagnosis was perithelioma. Patient recovered but with contraction of pupil and marked hoarseness. No recurrence was evident 6½ years later.

CASE 32. Binnie (14), 1906.

Clinical diagnosis, tuberculous adenitis; pathological, carotid tumor. Patient recovered and had no complications.

CASE 33. Coley (16), 1906.

Male, aged 53 years, had a tumor 6 years. The facial nerve was ligated. Pathological diagnosis was mixed cell sarcoma of the carotid gland. Patient recovered from operation but died from recurrence in 8 months.

CASE 34. Kuznetsoff (57), 1907.

Female, aged 48 years, had a tumor 2 years. The external carotid was ligated. No nerves were injured. Clinical diagnosis was cervical adenitis or carotid tumor. Recovery; no complications.

CASE 35. Rivet (78), 1907.

Female, aged 49 years, had a tumor 7 years. No vessels or nerves were ligated or injured. Clinical diagnosis was parotid or aberrant thyroid; pathological diagnosis was carotid tumor. Patient recovered but aphonia and difficulty in swallowing were present.

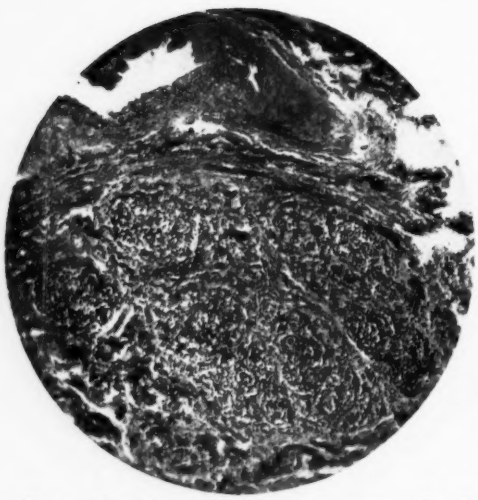

Fig. 3. Normal carotid body. Low power. By H. Klose (52).

Case 36. Cook (22), 1907.

Male, aged 32 years. All carotids and internal jugular were ligated. Clinical diagnosis was carotid aneurism. The pathological diagnosis was carotid tumor. Patient died 1 hour after operation from hæmorrhage.

Case 37. Boni (10), 1908.

Female, aged 34 years, had a tumor 4 years. All carotids and internal jugular were ligated. The vagus and sympathetic were injured. Clinical diagnosis was carotid tumor; pathological perithelioma, or adenoma of carotid gland. Recovery; no complications. No recurrence 2 years after operation.

Case 38. Cathcart (17), 1908.

Male, aged 33 years, had a tumor 2 years. All carotids were ligated. Pathological diagnosis was carotid tumor. Patient recovered and there was no recurrence 2 years later.

Case 39. Zondek (102), 1908.

Male, aged 63 years, had a tumor 9 years. All carotids were ligated, but no nerves were injured. No clinical diagnosis was made; pathological diagnosis, carotid tumor. Patient recovered.

Case 40. Licini (58), 1908.

Male, aged 28 years, had a tumor 9 years. No arteries or nerves were injured. Clinical diagnosis was carotid tumor; pathological, struma of carotid gland. Patient recovered.

Case 41. Green (39), 1908.

Female, aged 45 years, had a tumor 17 years. No arteries or nerves were injured. Tumor was not removed.

Case 42. Green (39), 1908.

Female, aged 7 years, had a tumor 2 years. All carotids were ligated. No nerves were injured. Patient recovered but had a permanent hemiplegia.

Case 43. Makara (65), 1908.

Male, aged 18 years, had a tumor 2 years. All carotids and the internal jugular were ligated. No nerves were injured. A clinical diagnosis of accessory thyroid was made but the pathological diagnosis was alveolar tumor of the carotid gland. Hemiplegia and death occurred 3 days after operation, of softening of the brain.

Case 44. Coley and Downs (27), 1909.

Male, aged 35 years, had a tumor 4 months. The external carotid and some branches were ligated. The pathological diagnosis was malignant carotid gland tumor. There was a prompt recurrence and death occurred in 4 months.

Case 45. Douglas (27), 1909.

Female, aged 23 years. No arteries or nerves were injured. A clinical diagnosis of tuberculous adenitis was made but pathological examination showed perithelioma. Patient recovered but had a temporary paralysis of the vocal cord.

Case 46. Anquez (3), 1909.

Female, aged 47 years, had a tumor 9 years. The common carotid, facial, and lingual arteries were ligated. No nerves were injured. No clinical diagnosis was made but the pathological diagnosis was perithelioma. Recovery.

Case 47. Lilienthal (60), 1909.

Female, aged 56 years, had a tumor 30 years. All carotids and the internal jugular were ligated. Clinical diagnosis was carotid tumor. Pathological diagnosis was malignant perithelial hæmangioma. Patient had a temporary aphasia and hemiplegia. She was cachectic for 3 years and death occurred 4 years after operation.

Case 48. Ligin (59), 1910.

All carotids were ligated but no nerve injury occurred. Pathological diagnosis was carotid gland. Recovery without complications.

Case 49. Mathews (68), 1911.

Male, aged 25 years, had a tumor 3 years. The external jugular was ligated. No clinical diagnosis was made but the operative diagnosis was carotid tumor. The tumor was not removed. Patient was reoperated upon by Dr. D. MacPherson, in 1913. He removed tumor of ear. Patient still alive in 1914.

Case 50. Woolley and Fee (101), 1912.

Female, aged 68 years, had a tumor 2 years. All carotids were ligated. No clinical diagnosis was made but the pathological diagnosis was sarcomatous degeneration of carotid tumor. Patient died of septicæmia 23 days after operation.

Case 51. Chiari (19), 1912.

Male, aged 39 years, had a tumor 3½ years. The external carotid and the internal jugular were ligated. The descendens hypoglossi was injured. Clinical diagnosis of carotid tumor was confirmed on pathological examination.

Case 52. Hollaender, 1912.

Female. No arteries or nerves were injured. Pathological diagnosis was carotid tumor. She recovered, but had a temporary, recurrent, laryngeal paralysis.

CASE 53. Randisi (75), 1912.

Female, aged 55 years, had a tumor 9 years. No arteries or nerves were injured. Clinical diagnosis was non-malignant tumor of the neck; pathological diagnosis, angiomyxoperithelioma. She recovered but had a right vocal cord paralysis.

CASE 54. Da Costa (16), 1913.

Female, aged 36 years, had a tumor 16 years. The external carotid was ligated. No nerves were injured. Clinical diagnosis was carotid tumor; pathological, perithelioma.

CASE 55. Sinyshin (9), 1913.

Female, aged 9 years. Pathological diagnosis, tumor of carotid gland.

CASE 56. Callison and Mackenty (9), 1913.

Male, aged 41 years, had a tumor 6 weeks. Biopsy was done first, then patient was re-operated upon for postoperative hæmorrhage and the common carotid was ligated. Clinical diagnosis was lymphosarcoma but the pathological diagnosis was endothelioma or perithelioma. Infection set in with postoperative hæmorrhage and hemiplegia. Death occurred 3 weeks after operation.

CASE 57. Graham (38), 1913.

Male, aged 27 years, had a tumor 7 years. The external carotid, facial arteries, and internal jugular were ligated. The vagus was injured. The clinical diagnosis was tumor of the tonsil but pathological examination disclosed perithelioma. Patient recovered but pupil was contracted and he was hoarse.

CASE 58. Reid (104), 1913.

Male, aged 46 years, had a tumor 5 years. The common carotid and the internal jugular were ligated. The vagus, and eleventh and twelfth nerves were injured. Clinical diagnosis was carotid aneurism but pathological diagnosis was adenoma of the carotid gland. Patient recovered.

CASE 59. Reid (104), 1913.

Male, aged 47 years, had a tumor 35 years. The internal jugular was ligated and the vagus was injured. No clinical diagnosis was made. The pathological diagnosis was carotid tumor. Death occurred on the operating table.

CASE 60. Levinsgs (4), 1913.

Male, aged 68 years, had a tumor 3 weeks. The internal jugular was ligated. No clinical diagnosis was made but the pathological diagnosis was carotid tumor. Recurrence took place in 4 months. Death.

CASE 61. Simmonds (88), 1913.

Male, aged 30 years. All carotids were ligated, no nerves were injured. No clinical diagnosis was made but the pathological diagnosis was paraganglioma intercaroticum. Hemiplegia and death followed operation.

CASE 62. Simmonds (88), 1913.

Female, aged 67 years. No arteries were ligated and no nerves were injured. No clinical diagnosis was made but the pathological diagnosis was paraganglioma intercaroticum. Patient recovered.

CASE 63. Neuber (72), 1913.

Female, aged 31 years, had a tumor 6 years. The common carotid was ligated and the recurrent laryngeal was injured. Clinical diagnosis was lipoma; pathological diagnosis, carotid tumor. Patient recovered but her voice was affected and she had paresis of the arm. There was no recurrence 4½ years after operation.

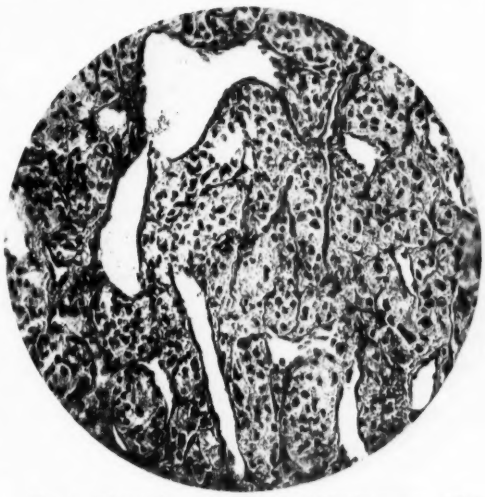

Fig. 4. Photomicrograph of carotid body tumor. High power.

CASE 64. Neuber (72), 1913.

Male, aged 30 years, had a tumor 9 years. All carotids were ligated. The vagus and sympathetic were injured. No clinical diagnosis was made but the pathological diagnosis was perithelioma. Death occurred 2 days after operation, of softening of brain.

CASE 65. Neuber (72), 1913.

Female, aged 67 years. No clinical diagnosis was made but the pathological diagnosis was perithelioma. Patient recovered.

CASE 66. Enderlen (29), 1913.

Male, aged 53 years. No ligations were done. No clinical diagnosis was made but the pathological diagnosis was tumor of the carotid gland. Patient recovered.

CASE 67. Schmidt (84), 1913.

Female, aged 52 years, had a tumor 20 years. The proximal end of the cut common carotid was sewed to the cut end of the internal carotid on the left side. Clinical diagnosis was tuberculous glands of the neck; pathological diagnosis, carotid tumor. Patient recovered.

CASE 68. Schmidt and Enderlen (84), 1913.

Female, aged 52 years, had a tumor 20 years (same patient as Case 67, but on opposite side of neck; right). No ligations were made or nerves injured. The clinical diagnosis was carotid tumor and the pathological diagnosis was the same. Patient recovered, but there was atrophy of the left half of the tongue. There was no recurrence for 1 year.

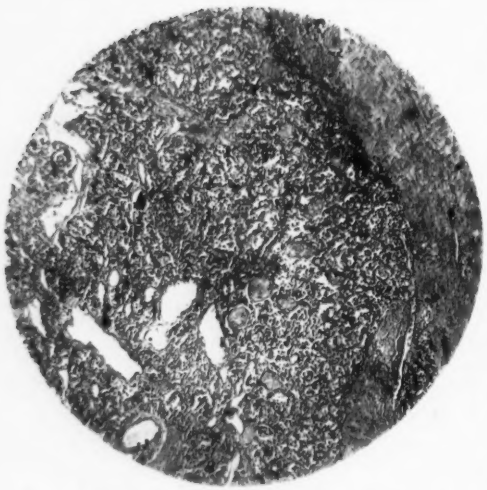

Fig. 5. Photomicrograph of carotid body tumor. Low power.

CASE 69. Groeneman (41), 1914.

Male, aged 42 years, had a tumor 2 years. The external and internal carotids were ligated; the vagus was injured. No clinical diagnosis was made but the pathological diagnosis was carotid tumor. Patient had a left hemiplegia. Death.

CASE 70. Balfour and Wildner (6), 1914.

Female, aged 34 years, had a tumor which had been present for some time. The internal jugular was ligated. Clinical diagnosis: aberrant thyroid probable. The pathological diagnosis was malignant tumor of the carotid gland. Hemiplegia appeared 10 days after operation. One year later there was a recurrence and the hemiplegia persisted.

CASE 71. Jopson and Kolmer (48), 1914.

Female, aged 27 years, had a tumor 2 months. The external carotid was ligated. No clinical diagnosis was made but the pathological diagnosis was perithelioma. Patient recovered and had no complications.

CASE 72. Russel (80), 1915.

Female, aged 25½ years, had a tumor for 11½ years. All carotids were ligated, but no nerves were injured. No clinical diagnosis was made but the pathological diagnosis was carotid tumor. Patient recovered but was slightly forgetful.

CASE 73. Collier (21), 1915.

Female, aged 50 years, had a tumor 5 years. The common carotid was ligated. No clinical diagnosis was made but the pathological diagnosis was perithelioma. Recovery followed operation but patient had paralysis of left vocal cord.

CASE 74. Steindl (92), 1915.

Female, aged 30 years, had a tumor 10 years. No ligations were made but the recurrent laryngeal and sympathetic nerves were injured. Clinical diagnosis was metastatic carcinoma of the tongue or tuberculous adenitis. The pathological diagnosis was carotid tumor. Patient recovered.

CASE 75. Owen (103), 1915.

Male, had a tumor 1 year. The external carotid was ligated. No nerves were injured. No clinical diagnosis was made but the pathological diagnosis was endothelioma of the carotid gland. Patient recovered and there were no complications.

CASE 76. Coughlin (14), 1915.

Male. All carotids and internal jugular were ligated. The vagus was injured. No clinical diagnosis was made but the pathological diagnosis was carotid tumor. Hemiplegia occurred and death took place 2 days after operation.

CASE 77. More (14), 1915.

A brother or sister of patient, Case 79, operated upon at the Mayo Clinic and the tumor was reported to be a carotid tumor.

CASE 78. Burge (14), 1916.

Male, aged 45 years, had a tumor 7 months. No ligations were made and no nerves were injured. Clinical diagnosis: tuberculous adenitis or carotid tumor. Pathological diagnosis; endothelial sarcoma of carotid gland. Recovery took place with slight deltoid paralysis.

CASE 79. More (14), 1915.

Female. No arteries or veins were ligated or nerves injured. Clinical diagnosis: carotid tumor or branchial cyst; pathological diagnosis: carotid tumor. Patient recovered.

CASE 80. Burge and Jepson (14), 1916.

Male, aged 42 years. No clinical diagnosis was made but the pathological diagnosis was endothelial sarcoma of the carotid gland. Patient recovered.

CASE 81. Schley (83), 1916.

Male, aged 37 years, had a tumor 20 years. All carotids were ligated but no nerves were injured. No clinical diagnosis was made but the pathological diagnosis was carotid gland tumor. Patient recovered but there was temporary hoarseness.

CASE 82. Shipley and Lynn (87), 1916.

Female, aged 16 years, had a tumor 2 months. All carotids and internal jugular were ligated. Clinical diagnosis, sarcoma; pathological diagnosis, carotid tumor. Patient recovered. She had a rapid pulse for 5 days after operation. There was no recurrence 5 years after operation.

CASE 83. Morestin (9), 1916.

Male, aged 46 years, had a tumor 15 years. No ligations were made or nerves injured. No clinical diagnosis was made but the pathological diagnosis was paraganglioma. Recovery.

CASE 84. Winslow (100), 1916.

Male, aged 24 years, had a tumor 8 years. Common and internal carotid were ligated. No nerves were injured. Clinical diagnosis was carotid tumor and pathological diagnosis, perithelioma. Patient recovered but voice was affected. No recurrence was apparent 1½ years after operation.

CASE 85. Cahill and Taylor (15), 1917.

Female, aged 51 years, had a tumor 8 years. The internal jugular was ligated and no nerves were injured.

CASE 86. Gay (62), 1888.

Female, aged 24 years, had a tumor 2 years (left side). No ligations were made or nerves injured. Clinical diagnosis, tumor of neck. Recovery.

CASE 87. Lund (62), 1917.

Female, aged 54 years, had a tumor 16 years (right side—this is same patient as Case 86). All carotids were ligated but no nerves were injured. Clinical diagnosis was carotid tumor and was confirmed by pathological examination. Patient recovered. Twenty-nine years before she had had left sided tumor removed.

CASE 88. Groenberger (40), 1917.

Male, aged 67 years. It was impossible to remove the tumor because it had extended into the mediastinum. Laminectomy was done for pain. Pathological diagnosis, perithelioma. Death occurred 3 days after operation.

CASE 89. Wetterdal (52), 1917.

Male, aged 47 years, had tumor 6 months. The tumor infiltrated the vessels, hence it was only partially removed. Pathological diagnosis, carotid tumor. Death occurred 12 days after operation.

CASE 90. Wetterdal (52), 1917.

Female, aged 56 years. No ligations were done and the nerves were not injured. Clinical diagnosis was tumor of the neck; pathological diagnosis, carotid tumor. Recovery.

CASE 91. Fowler (33), 1917.

Female, aged 58 years, had tumor 2 years. All carotids were ligated. The vagus, hypoglossus, and recurrent laryngeal nerves were injured. No clinical diagnosis was made; pathological diagnosis was malignant carotid tumor with tendency to endothelial type. Patient recovered, but was hoarse and had a cough.

CASE 92. Cohn (20), 1918.

Male. The internal jugular was ligated. The common carotid was only a fibrous cord. The clinical diagnosis was carotid tumor or lymphosarcoma; pathological diagnosis, carotid tumor. Death ensued in 10 days.

CASE 93. Wiener (99), 1918.

Female, aged 37 years, had tumor 8 years. All carotids and internal jugular were ligated. No clinical diagnosis was made but pathological diagnosis was perithelioma. Patient recovered. Patient had a partial right hemiplegia, right facial paresis, and also paralysis of vocal cord. He was given X-ray treatment after operation. No recurrence was noted 4 months later.

CASE 94. Reenstjerna (77), 1919.

Female, aged 36 years, had tumor 11 years. The external carotid was ligated, and the recurrent laryngeal was injured. No clinical diagnosis was made but the pathological diagnosis was carotid tumor. Patient recovered, but there was paralysis of the vocal cord.

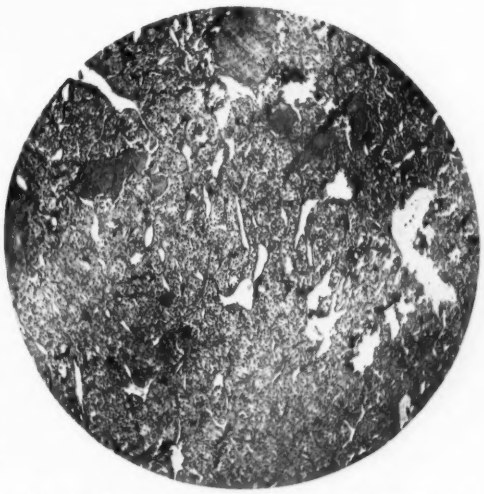

Fig. 6. Photomicrograph of carotid body tumor. Low power.

CASE 95. Reenstjerna (77), 1919.

Male, aged 43 years, had tumor 3 years. All carotids were ligated. No clinical diagnosis was made but the pathological diagnosis was carotid tumor. Patient died of hemiplegia 4 days after operation.

CASE 96. Reid (104), 1919.

Female, aged 37 years, had tumor 4 years. All carotids and the internal jugular were ligated. The vagus and hypoglossus were injured. No clinical diagnosis was made but the pathological diagnosis was round cell sarcoma of carotid gland. Treatment with radium had no effect; operation resulted in recovery with paralysis of right vocal cord and deviation of tongue.

CASE 97. Thompson (95), 1919.

Male, aged 56 years, had tumor 20 years. All carotids and the internal jugular were ligated. No nerves were injured. Clinical diagnosis was carotid tumor or aneurism; pathological diagnosis, perithelioma. Patient recovered and there were no complications.

CASE 98. Davis and Traut (96), 1920.

Male, aged 52 years, had tumor 3 years. No ligations were made but the descendens hypoglossi was injured. No clinical diagnosis was made; pathological diagnosis was carotid tumor. Hemiplegia appeared one day after operation and patient died in 5 days of bronchopneumonia.

CASE 99. Keynes (51), 1921.

Female, aged 31 years, had tumor 2 years. Clinical diagnosis was carotid tumor; pathological diagnosis, endothelioma of carotid gland. Patient recovered but had a temporary paralysis of the orbicularis oris.

CASE 100. Birman (9), 1922.

Male, aged 54 years, had tumor 5 years. All carotids were ligated; no nerves were injured.

Clinical diagnosis was lymphoma but pathological examination disclosed carotid tumor.

CASE 101. Klose (52), 1922.

Female, aged 30 years, had tumor 8 years. The common carotid and internal jugular were ligated. The descendens hypoglossi and superior laryngeal were injured. Clinical diagnosis was carotid tumor and pathological examination confirmed this. Patient recovered.

CASE 102. Klose (52), 1922.

Male, aged 60 years, had tumor 21 years. Clinical diagnosis was carotid tumor. He was not operated upon. An attempt to remove the tumor in 1902 was prevented by hæmorrhage.

CASE 103. Descarpentries (79), 1922.

Female, aged 38 years, had tumor 10 years. All carotids were ligated. No nerves were injured. Clinical diagnosis was adenitis; pathological diagnosis, non-malignant carotid tumor. Patient recovered and recovery was permanent.

CASE 104. Edye (79), 1922.

Male, aged 27 years. All carotids were ligated and the vagus was injured. Clinical diagnosis was cervical tumor; pathological diagnosis, carotid tumor. Recovery followed operation but paresis of one-half tongue and hoarseness appeared. No recurrence was evident 8 years after operation.

CASE 105. Edye (79), 1922.

Male, aged 38 years, had tumor 2 months. All carotids and internal jugular were ligated. The vagus was injured. No clinical diagnosis was made; pathological diagnosis, carotid tumor. Recovery followed operation but left vocal cord paralysis appeared. No recurrence was evident 6 months after operation.

CASE 106. Fedeii (4), 1922.

Male, aged 59 years, had tumor 2 years. No ligations were made and no nerves were injured. Clinical diagnosis of carotid tumor was confirmed. Patient recovered without complications.

CASE 107. Miller and Garland (70), 1923.

Female, aged 35 years, had tumor 4 years. The common and external carotid arteries were ligated and no nerves were injured. Clinical diagnosis was tuberculous adenitis; pathological diagnosis, perithelioma. Patient recovered but with a temporary paralysis of one-half of the tongue. No recurrence appeared in one year.

CASE 108. Knighton (53), 1923.

Male, aged 58 years, had tumor 15 years. The external carotid was ligated. Clinical diagnosis was tumor of neck (had fainting attacks from pressure on vagus). Pathological diagnosis was non-malignant carotid tumor. Recovery with complete aphonia followed operation. No recurrence was evident in one year.

CASE 109. Boat (12), 1923.

Male, aged 42 years, had tumor 6 months. All carotids and the internal jugular were ligated. No nerves were injured. Clinical diagnosis was extrinsic tumor of larynx with Stokes-Adams disease; Pathological diagnosis, carcinoma (?) of carotid gland. Patient died one week after operation of acute œdema of the larynx.

CASE 110. Bérard and Dunet (8), 1923.

Patient had tumor 3 years. The external carotid and the internal jugular were ligated. The vagus was injured. Clinical diagnosis was branchiogenetic carcinoma, but pathological diagnosis was malignant carotid tumor. Aphasia and hemiplegia (internal carotid thrombosis) developed and death ensued 2 days after operation.

CASE 111. Guthrie (42), 1924.

Female, 39 years, had tumor 3 years. Clinical diagnosis was mixed tumor of parotid but operative diagnosis was adenoma of the carotid gland. Tumor was not removed.

CASE 112. Pinker (9), 1924.

Common carotid and internal jugular were ligated. The vagus was injured. No clinical diagnosis was made; pathological diagnosis was carotid tumor. Recovery.

CASE 113. Stiltmann (9), 1924.

Patient had tumor 9 years. No clinical diagnosis was made but pathological diagnosis showed carotid tumor.

CASE 114. Thorn (9), 1924.

Patient had tumor 4 years. The common carotid was ligated. No clinical diagnosis was made but the pathological diagnosis was carotid tumor. Death occurred 24 hours after operation, of softening of the brain.

CASE 115. Harttung (43), 1924.

Male, aged 42 years, had tumor 4 years. The internal jugular was ligated but no nerves were injured. Clinical diagnosis was malignant tumor of the neck; pathological diagnosis, carotid tumor. Patient recovered without complications.

CASE 116. Royster (79), 1924.

Female, aged 27 years, had tumor several years. All carotids were ligated. No nerves were injured. Clinical diagnosis was tuberculous adenitis (?). Pathological diagnosis was perithelioma. Recovery without complications ensued. No recurrence appeared in 3 years.

CASE 117. Abe (1), 1924.

Patient, aged 26 years. Clinical diagnosis was egg-sized tumor of neck; pathological diagnosis, tumor of parenchyma.

CASE 118. Jassowicz (47), 1924. Unable to obtain journal.

CASE 119. Sullivan and Fraser (94), 1925.

Female, aged 49 years, had tumor 10 years. No ligations were made and no nerves were injured. Clinical diagnosis was cervical adenitis or carotid tumor; pathological diagnosis, perithelioma. Patient recovered, and had no recurrence in 1½ years.

CASE 120. Sullivan and Fraser (94), 1925.

Male, aged 38 years, had tumor 8 years. No ligations were made. Clinical diagnosis was carotid tumor or aberrant thyroid. Pathological diagnosis was neuroblastoma of carotid gland. Patient recovered. Contraction of pupil persisted 21 months after operation.

CASE 121. Sheehan and Rabiner (86), 1925.

Female, aged 50 years, had tumor 2 years. No ligations were made and no nerves were injured. Clinical diagnosis was aneurism of the facial artery. Pathological diagnosis was carotid tumor. Patient recovered and was treated with X-ray.

CASE 122. Maiocchi (4), 1925.

Male, aged 16 years, had tumor 2 years. No ligations were made and no nerves were injured. Clinical diagnosis was not made but the pathological diagnosis was fibro-angioma of the carotid gland. Patient recovered but had a temporary paralysis of the cervical sympathetic.

CASE 123. Labey (4), 1925.

Female, aged 65 years. The external carotid was ligated but no nerves were injured. No diagnosis was reported. Hemiplegia followed operation and patient died in 3 days.

CASE 124. Leclerc (4), 1925.

Female, aged 37 years, had tumor 7 years. All carotids were ligated; no nerves were injured. No clinical diagnosis was made; pathological diagnosis was perithelioma. Patient recovered, but suffered from aphonia and dysphagia for 2 years. There was no recurrence.

CASE 125. Bonikowsky (11), 1926.

Male, aged 19 years, had tumor 2 years. No ligations were done and no nerves were injured. Clinical diagnosis was cyst or fascial sarcoma. Pathological diagnosis was carotid tumor. Hemiplegia developed 3 days after operation (embolus), but patient recovered.

CASE 126. Aperlo and Rossi (4), 1926.

Male, aged 64 years, had tumor 9 months. No arteries were ligated and no nerves were injured. Clinical diagnosis was possible carotid tumor; and pathological diagnosis, paraganglioma (mixed tumor of carotid gland). Patient recovered but there were no complications.

CASE 127. Holoway (4), 1926.

Female, aged 35 years. No arteries were ligated. Hæmorrhage into the tumor was aspirated. No nerves were injured. No clinical or pathological diagnosis was reported. Recovery.

CASE 128. Bowen and Miller (13), 1926.

Female, aged 36 years, had tumor 3 years. No arteries were ligated and no nerves were injured. No clinical diagnosis was made but pathological diagnosis showed adenoma of carotid gland. Patient recovered but was hoarse for months.

CASE 129. Traut (96), 1927.

Female, aged 46 years, had tumor 12 years. All carotids were ligated. The vagus was injured. Clinical diagnosis was tuberculous adenitis; pathological diagnosis, carotid tumor. One day after operation hemiplegia and aphasia were noticed. Death occurred 22 days after operation from bronchopneumonia.

CASE 130. Duroux and Rollin (28), 1927.

Male, aged 48 years, had tumor 5 months. No ligations were made (this case was curetted out). No nerves were injured. No clinical diagnosis was made but pathological diagnosis was malignant tumor of the carotid gland. Patient recovered. Death occurred 7 months later from recurrence and general cachexia.

CASE 131. Floercken (32), 1927.

Female, aged 34 years, had tumor 10 years. The common carotid, external maxillary, and internal jugular were ligated.

CASE 132. Fitzgerald (31), 1927. Unable to get journal.

CASE 133. Harvey (44), 1927.

Female, aged 32 years, had tumor 1 year. No vessels were ligated and no nerves were injured. No clinical diagnosis was made but the pathologic diagnosis was benign hæmorrhagic cyst of the carotid gland. Patient recovered without complications.

CASE 134. Bevan and McCarthy, 1928.

Male, aged 39 years, had tumor 1½ years. No vessels were ligated and no nerves were injured. The tumor was not removed. Clinical diagnosis was probable tuberculous adenitis. Operative diagnosis was carotid tumor. Patient was treated with X-rays.

POST-MORTEM CASES

1. Leithoff (9), 1904.

Patient had a cherry-sized tumor of the neck which pathologically was carotid tumor.

2. Oberdorfer (9), 1905.

Female, aged 58 years. Pathological diagnosis was hamartoma of carotid gland.

3. Hedinger (9), 1905.

Female, aged 62 years. Pathologic diagnosis was carotid tumor.

4. Cecca (18), 1906.

Male, aged 40 years, had tumor 4 years. Pathologic diagnosis was angiosarcoma of carotid gland.

5. Beitzke (7), 1909.

Female, aged 56 years. Pathologic diagnosis was struma intercarotica.

6. Alezais and Peyron (2), 1910.

Adult male. Pathological diagnosis was malignant carotid gland tumor, paraganglioma.

7. Alezais and Peyron (4), 1911.

Pathologic diagnosis was paraganglioma.

8. Alezais and Peyron (4), 1911.

Pathologic diagnosis was carotid tumor.

9. Sapegno (81), 1913.

Female, aged 64 years, had tumor 1 year. Patient died of bronchopneumonia while in the hospital awaiting operation. Pathologic diagnosis was carotid tumor.

BIBLIOGRAPHY

1. ABE. Ganu, 1924, xviii, 80–81.
2. ALEZAIS and PEYRON. Paragangliome carotidien à évolution. Bull. de l'Ass. franç. p. l'étude du cancer, 1910, iii, 181–190; Ztschr. f. Chir., 1911, xxxviii, 1107.
3. ANQUEZ. A general review of neoplasms of the carotid gland; with report of a case. Internat. Clin., 1920, iii, 208–228.

4. APERLO, G., and ROSSI, F. I tumori della ghiandola Carotidea. Clin. Chir., Milano, 1927, January 26, 139.
5. ARNOLD. Ueber die struktur des Ganglion intercaroticum. Arch. f. path. Anat., 1865, p. 190.
6. BALFOUR, D. C., and WILDNER, FRANZ. The intercarotid paraganglion and its tumors. Surg., Gynec. & Obst., 1914, xviii, 203–213.
7. BEITZKE. Ueber einen Tumor der Carotisdruese. Charité-Ann., Berl., 1909, xxxiii, 382–389.
8. BÉRARD and DUNET. Tumeur de la glande carotidienne. Presse méd., 1923, March.
9. BIRMAN. Ueber Strumen der Carotisdruese. Deutsche Ztschr. f. Chir., 1924, clxxxvi, 384–402.
10. BONI, E. Contributio allo studio dei tumori del tuberculo carotidea. Pensiero med., 1912, ii, 761, 781.
11. BONIKOWSKY, H. Struma von der Carotisdruese. Zentralbl. f. Chir., 1926, liii, 2455–2457.
12. BOOT, G. W. Tumor of the carotid gland with Stokes-Adams syndrome. Ann. Otol., Rhinol. & Laryngol., 1923, xxxii, 1241–1245.
13. BOWEN, W. F., and MILLER, M. B. Tumor of carotid body; report of a case. J. Kansas M. Soc., 1926, xxvi, 321.
14. BURGE, A. J. Report of two cases of malignant carotid gland tumors. Tr. West. Surg. Ass., 1916, xxvi, 319–322.
15. CAHILL, G. F., and TAYLOR, R. M. A case of tumor of the carotid body. J. Am. M. Ass., 1917, lxviii, 1898–1899.
16. CALLISON, J. G., and MACKENTY, J. E. Tumors of the carotid body. Ann. Surg., 1913, lviii, 740–765.
17. CATHCART, R. S. Tumors of carotid gland; with report of a case. J. South Carolina M. Ass., 1908, iv, 304.
18. CECCA, R. Sulla patalogia del corpocuscolo intercarotide. Clin. chir., 1906, xiii, 1177–1184.
19. CHIARI, O. M. Ueber einen Fall von Carotisdruesentumor. Beitr. z. klin. Chir., 1912 (v. Hacker's Festschrift), lxxxi, 599–608.
20. COHN, S. Tumor of the carotid body. New Orleans M. & S. J., 1918, lxx, 658.
21. COLLIER, C. Tumor of the carotid body; report of a case. Surg., Gynec. & Obst., 1915, xx, 484.
22. COOK, J. E. Case of tumor of carotid body. Surg., Gynec. & Obst., 1907, v, 342.
23. CUNÉO, M. M., and DAINVILLE. Fibrome volumineux du con ayant determine de phénomènes de Compression nerveuse du grande sympathique et du facial. Soc. Anatomique, 1903, July 10.
24. DACOSTA, J. C. Report of a case of tumor of the carotid body. Ann. Surg., 1906, xliv, 393–403.
25. DEITERICH. Epithelkoerperchen und Carotisdruese. Beitr. z. klin. Chir., 1924, cxxxi, 708.
26. DOBROMIDOFF. Zur Pathologie und Therapie der Tumoren des Glomus caroticum. Chirurgica, 1908, nr, 135; Zentralbl. f. Chir., 1908, xxxv, 956.
27. DOUGLAS, J. Perithelioma of the carotid gland. Med. Rec., 1909, lxxv, 397.
28. DUROUX, E., and ROLLIN, L. Case report. Bull. méd., Par., 1927, xli, 1107.
29. ENDERLEN. Beitr. z. klin. Chir., 1913, lxxxiii, 726.
30. FISCHER. Ueber die Funktion der Carotisdruese. Ztschr. f. d. ges. exper. Med., 1924, xxxix, 477.
31. FITZGERALD. St. Barth. Hosp. J., 1927, xxxiv, 62.
32. FLOERCHEN, H. Ein Beitrag zu den Tumoren der Carotisdruese. Muenchen. med. Wchnschr., 1927, lxxiv, 931.
33. FOWLER, ROYALE H. Sarcoma of intercarotid body. Med. Times, 1917, xlv, 341–342.
34. FRUGONI, C. Études sur la Gland Carotidienne de Luschka. Arch. ital. de biol., 1915, lix, 208–212.
35. FUNKE, J., and HEARN. The carotid body; anatomy, histology, embryology, and tumors arising from it. Am. Med., 1904, viii, 109–115.
36. GILFORD, H., and DAVIS. Potato tumors of the neck and their origin as endothelioma of the carotid body; with an account of three cases. Practitioner, 1904, lxxiii, 729–739.
37. GOMEZ, L. P. The anatomy and pathology of the carotid gland. Am. J. M. Sc., 1908, cxxxvi, 98–110.
38. GRAHAM, A. Tumors of the carotid body; with report of two cases. Cleveland M. J., 1913, vii, 537–549.
39. GREEN. Tumors of the carotid gland; two cases. Surg., Gynec. & Obst., 1908, xxx, 477–480; J. Am. Inst. Homœop., 1909, i, 55–57.
40. GROENBERGER, G. Ein Fall von Carotisdruesentumor. Hygea, 1917, xxix, 4; Zentralbl. f. Chir., 1917, 48.
41. GROENEMAN, W. Zur Kasuistir der Geschwuelste des Nodulus Caroticus. Arch. f. path. Anat., 1914, ccxviii, 163–170.
42. GUTHRIE, D. Tumor of carotid body. J. Laryngol. & Otol., 1924, xxxix, 635.
43. HARTTUNG. Carotisdruesentumor. Beitr. z. klin. Chir., 1924, cxxxi, 142–149.
44. HARVEY, FRANK. A tumor of the carotid body. Brit. J. Surg., 1928, xvi, 163–165.
45. HEINLETH. Beitrag. zur Histologie der Perithelioma glandulae caroticae. Zentralbl. f. allg. Path. u. path. Anat., 1900, xi, 559–603; Ein Fall von Carotisdruesenperithelioma. Muenchen. med. Wchnschr., 1900, xlvii, 899–902.
46. HOLLAENDER. Tumor der Glandula carotica. Verhandl. d. Berl. med. Gesellsch., 1912–1913, xliii, 167; Berl. klin. Wchnschr., 1912, xlix, 1150.
47. JASSOWICZ, W. K. Turkestanski M. J., 1924, 3.
48. JOPSON, J. H., and KOLMER, J. Tumor of carotid body. Tr. Phila. Acad. Surg., 1915, xvii, 137–140; Ann. Surg., 1914, lx, 789–792.
49. KAUFMANN, E., and RUPPANNER: Ueber die alveolaeren Geschwuelste der Glandula carotica. Tsch. Ztsch. f. Chir., 1905, lxxx, 259–319.
50. KEEN, W. W., and FUNK, J. Tumors of the carotid gland. J. Am. M. Ass., 1906, lvii, 469–566.
51. KEYNES, GEOFFREY. A case of tumor of the carotid body. Brit. J. Surg., 1921–22, ix, 159–162.
52. KLOSE, H. Beitraege zur Chirurgie der sog. Carotisdruese. Arch. f. klin. Chir., 1922, cxxi, 689–724.
53. KNIGHTON, J. E. Case report of tumor involving carotid sheath, presenting some interesting diagnostic problems. New Orleans M. & S. J., 1924, lxxvii, 237.
54. KOHN, A. Ueber den Bau und die Entwicklung der sog. Carotisdruese. Arch. f. mikr. Anat., 1900, lvi, 81–148.
55. KOPFSTEIN, W. Beitrag zur Kenntnis der Geschwuelste der Carotisdruese. Wien. klin. Rundschau., 1895, ix, 82–97; Ein weiterer Beitrag zur Kenntnis der intercarotischen Geschwuelste. Wien. klin. Rundschau., 1895, xxii, 23.
56. KRETSCHMAR. Ueber eine Geschwuelst der Gland carotica. Inaugural dissertation, Geissen, 1893.
57. KUSNETSOFF. Tumors of the carotid gland and their surgical treatment. Khirurg. Arkh. Velyaminova, 1910, xxvi, 25–29.
58. LICINI, C. Ueber einen Tumor der Glandula carotica. Deutsche Ztschr. f. Chir., 1908, xcvi, 327–339.
59. LIGIN. After Kusnetsoff from Callison and MacKenty.
60. LILIENTHAL, H. Endothelioma of the carotid body. Ann. Surg., 1909, i, 808.

61. Idem. Tumor of the carotid body. Ann. Surg., 1914, lix, 132–134.
62. LUND, F. B. A case of bilateral tumor of the carotid body. Boston M. & S. J., 1917, clxxvi, 621–623.
63. LUSCHKA. Ueber de druesenartige Natur des sog. Ganglion intercaroticum. Arch. f. Anat. u. Physiol., 1862.
64. MACPHATER, N. Removal of internal jugular vein and carotid arteries in order to remove a tumor in the left side of the neck. New York & Phila. M. J., 1903, lxxviii, 157.
65. MAKARA. Ueber die Geschwuelste de Glandula carotica. Orvosi Hetilap, 1907; Zentralbl. f. Chir., 1908, xxxv, 27.
66. MALINOWSKY. Contribution à l'étude des tumeurs de la glande carotidienne, Kiev, 1899.
67. MARCHAND, F. Beitraege zur Kenntnis der normalen und pathologischen Anatomie der Glandula carotica und der Nebennieren. Festschr. f. Rudolph Virchow, 1891, i, 535.
68. MATHEWS, F. S. Tumors of the carotid. Ann. Surg., 1914, lix, 752; Personal communication to Callison and Mackenty.
69. MIDDLETON and BIERRING. Tr. Iowa State M. Soc., 1897, xv, 94.
70. MILLER, R. H., and GARLAND, F. E. Tumor of the carotid gland. Boston M. & S. J., 1923, cxci, 659–662.
71. MOENCKEBERG, J. G. Die Tumoren der Glandula carotica. Beitr. z. path. Anat. u. z. allg. Path. 1905, xxxviii, 1–66.
72. NEUBER, C. E. L. Ueber Geschwuelste der Carotisdruese. Arch. f. klin. Chir., 1913, cii, 289–296.
73. PALTAUF, R. Ueber Geschwuelste de Glandula Carotica, nebst linen Beitraege zur Histologie und Entwicklungsgeschichte derselben. Beitr. z. path. Anat. u. z. allg. Path., 1891–92, xi, 260–301.
74. PAUNZ. Handbuch der Spez. Path. Anat. und Hist., Henke-Lubarsch, viii, 1060–71.
75. RANDISI, F. Di un tumore vascolare della ghiandola carotidea. Clin. chir., 1912, xx, 228–238.
76. RECLUS and CHEVASSU. Les tumeurs du corpuscle rétro-carotidien. Rev. de chir., 1903, xxviii, 149.
77. REENSTJERNA, J. A contribution to the knowledge of the carotid gland. Arch. f. inn. Med., 1919, ci, 215–226.
78. RIVET. Tumeur Corpuscle Carotidien. Gaz. méd. de Nantes, 1907, xxv, 626–628.
79. ROYSTER, H. A. Tumors of the carotid body. South. M. J., 1924, xvii, 196.
80. RUSSEL, C. W. Tumors of the carotid body; with report of a case. J. Missouri State M. Ass., 1915, xii, 521–523.
81. SAPEGNO. Tumore maligno del paraganglio carotideo. Arch. sc. med., Torino, 1913, xxxvii, 367–403.
82. SCHAPER. Beitr. zur Histol. der Glandula carotica. Arch. f. mikr. Anat., 1892, p. 287.
83. SCHLEY, W. S. Tumor of the carotid body; excision with ligature of the carotid arteries. Ann. Surg., 1917, lxvi, 252–254.
84. SCHMIDT, J. E. Beitraege zur Kenntnis der Glandula carotica und ihrer Tumoren. Beitr. z. klin. Chir., 1913, lxxxvii, 301–309.
85. SCUDDER, L. C. Tumor of intercarotid body; a report of one case together with all cases in literature. Am. J. M. Sc., 1903, vi, 384–389.
86. SHEEHAN, J. E., and RABINER, MAX. Report of an unusual case of tumor of the carotid body. Laryngoscope, 1927, xxxvii, 433.
87. SHIPLEY, A. M., and LYNN, FRANK S. Carotid tumor and aneurism of the internal carotid; reports of cases. J. Am. M. Ass., 1916, lxvi, 1602–1604.
88. SIMMONDS. Ueber Geschwuelste der Carotisdruese. Muenchen. med. Wchnschr., 1913, xxxix, 1337.
89. SINIUOUSCHINE, N. V. Tumeur dévéloppée eux dépens de la glande carotidienne. Soc. de Cr. de Moscow, 1901.
90. SINYSHIN, N. V. Tumor of the carotid gland. Med. Obozr., 1913, lxxix, 34–39; Deutsche Ztschr. f. Chir., 1924, clxxxvi, 398.
91. SMITH, CHRISTIANNA. The origin and development of the carotid body. Am. J. of Anat., 1924–25, xxxiv, 87–125.
92. STEINDL. Beitrag. zur Kenntnis der Carotisdruesen geschwuelste. Deutsche Ztschr. f. Chir., 1915, cxxxii, 1–21.
93. STILLING. Anat. Anz., 1898, 15.
94. SULLIVAN, R. P., and FRASER, ALEX. Tumors of the carotid body. Surg., Gynec. & Obst., 1927, xlv, 209.
95. THOMPSON, J. O. Perithelioma of ganglion intercaroticum. China M. J., 1920, xxxiv, 32–33.
96. TRAUT, E. F. Tumor of carotid body. Surg., Gynec. & Obst., 1927, xlv, 829–832.
97. WALDEYER. Arch. f. path. Anat., 1872, p. 55.
98. WETTERDAL, P. Beitrag till Rannedomen om Karotiskörtel tumören. Hygiea, 1916, p, 1761; Klose, Arch. f. klin. Chir., 1922, cxxi, 689–724.
99. WIENER, J. Excision of tumor of the carotid body. Ann. Surg., 1919, lxix, 648–650.
100. WINSLOW, P. Tumors of the carotid body. Tr. Am. Surg. Ass., 1916, 385–403; Ann. Surg., 1916, lxiv, 257–267.
101. WOOLLEY, P. G., and FEE. An alveolar tumor of the carotid gland with sarcomatous transformation. Johns Hopkins Hosp. Bull., 1912, xxiii, 146–151.
102. ZONDECK, M. Ein tumor der Glandula carotica. Berl. klin. Wchnschr., 1908, xlv, 219–222.
103. OWEN, L. J. Endothelioma of intercarotid body. Ann. Surg., 1915, lxi, 382.
104. REID, M. R. Adenomata of the carotid gland. Johns Hopkins Hosp. Bull., 1920, xxxi, 177–184.
105. MORESTIN, H. Tumeur du Corpuscule inter-ou retro-carotidien. Bull. mém. Soc. de chir. de Par., 1916, xlii, 2308–2315.

PATHOLOGICAL LESIONS OF THE GALL BLADDER[1]

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THE gall bladder, because of its accessibility and the frequency with which it is removed, readily affords opportunity for the grouping and the correlation of clinical and pathological data.

I have prepared a simple grouping of diseases of the gall bladder based chiefly on the gross examination of 4,575 specimens removed at operation. Microscopic examinations were made of about 50 of each of the larger groups, and of all of the smaller groups. Twenty-five to 50 histories of each group were studied and comparisons made.

One specimen may show more than one type of pathological change. I have, therefore, grouped these specimens in sequence according to the amount of pathological change shown. The grouping is as follows: (1) chronic catarrhal cholecystitis; (2) "strawberry gall bladder" or "cholesterosis"; (3) chronic fibrcus cholecystitis; (4) subacute and acute cholecystitis; (5) empyema of the gall bladder; (6) gangrene of the gall bladder; (7) hydrops of the gall bladder; (8) papilloma of the gall bladder; (9) adenoma of the gall bladder; and (10) malignancy of the gall bladder (tabulation).

CHRONIC CATARRHAL CHOLECYSTITIS

In this group are included gall bladders showing only slight pathological change (Fig. 1), such as slight change in color and thickening of the walls. It is this type of gall bladder which is frequently removed, not so much because of gross change in the organ itself, as because of a definite history of cholecystic disease and of associated conditions, such as a round-edged liver, a surrounding area of hepatitis, or the presence of a sentinal lymph node, as stressed by C. H. Mayo. Microscopic examination of this type of gall bladder reveals varying degrees of œdema and congestion of the villi and lymphocytic infiltration.

In the 2,051 cases (44.8 per cent) in this group stones were present in 1,250 (61 per cent) and absent in 801 (39 per cent). In 481 of the 801 specimens in which stones were not found, there was slight thickening and change in color. Of the 1,250 specimens with stones, 436 showed only minimal gross evidence of disease. It is possible, as Moynihan (1928) and Judd (1925) have stated, that this type of gall bladder assumes a normal appearance between attacks, not unlike that of the "interval" appendix. This is substantiated by the frequent occurrence of stones in an otherwise normal appearing gall bladder. The histories of 50 patients were studied to determine the clinical manifestations. The symptoms were those of gastric disturbance. The chief complaints were gastric distress, nausea, vomiting, belching, regurgitation, flatulence, constipation, and varying degrees of pain, the severity of which was evidently dependent on the amount of infection or the presence of calculi. The average age of the patients was 44 years, of the youngest 27, and of the oldest 67. Sixty-one per cent were women and 39 per cent were men. On examination, tenderness was elicited in the right upper quadrant in 74 per cent. The average weight was 150 pounds. The gastric contents of 32 patients showed an average total acidity of 52 and an average free hydrochloric acid value of 32 in terms of tenth normal sodium hydroxide; achlorhydria occurred in 3 cases.

"STRAWBERRY GALL BLADDER" OR "CHOLESTEROSIS"

Strawberry gall bladder, or cholesterosis, is a condition in which lipoid deposit is found embedded in the epithelial cells or stroma, thus giving the characteristic strawberry appearance (Fig. 2). Moynihan (1909) described this condition but at that time thought it was due to fine granules of calculus embedded in the mucosa. MacCarty (1910) described the condition in detail and named it strawberry gall bladder. He first believed it to be secondary to sloughing of the superficial epithelial cells and bile-stained stroma, but in

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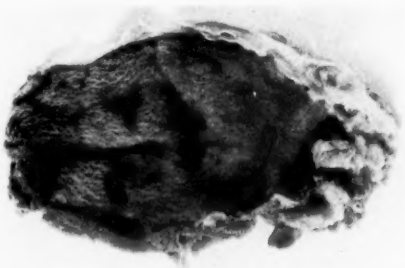

Fig. 1. Chronic catarrhal cholecystitis; slight thickening of the walls is shown.

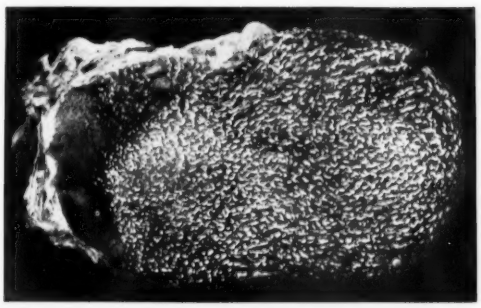

Fig. 2. "Strawberry gall bladder" or "cholesterosis."

1919 he described it as being due to a lipoid deposit within the swollen connective tissue which lies just beneath the epithelium of the villi. Boyd (1923) showed that the lipoid was a cholesterol-ester, and Mentzer (1925) applied the name cholesterosis to the condition. Various experiments have been carried out to determine whether the deposit of cholesterol is a result of excretory or secretory function of the gall bladder, and from these it appears obviously due to a secretory function. MacCarty (1919) reported that 18 per cent of 4,998 surgically removed gall bladders showed deposits of cholesterol. Mentzer (1926), in a series of 612 necropsies on adults, reported cholesterosis in 21 per cent.

The incidence in my series is about the same as that in the series which Mentzer reported. There were 1,094 cases (23 per cent). Of these, 552 (50 per cent) were associated with stones. In only 465 (43 per cent) were the walls definitely thickened. Papillomata were present in 164 (14 per cent).

The microscopic picture in cholesterosis has been described by MacCarty (1919), Corkery (1922), and Boyd (1923). The lipoid is usually deposited as fine granules in the cells, lumen, and acini, just under the base of the epithelial cells and in the cells of the submucosa (Fig. 3). Occasionally the lipoid occurs in masses. Varying grades of round cell infiltration also occur.

I studied the histories of 50 patients with this type of cholecystic disease but I could find nothing which might distinguish this type from chronic catarrhal cholecystitis. Of these 50 patients 69 per cent were women and 31 per cent were men. The average duration of symptoms was 6 years. The average weight was 153 pounds. Some of these patients, who were of normal weight at the time of examination, had been definitely overweight. Sixteen patients had had test-meals. The average total acidity was 54 and the average free hydrochloric acid value was 33; two had achlorhydria.

CHRONIC FIBROUS CHOLECYSTITIS

Chronic fibrous cholecystitis is a condition of the gall bladder in which there is marked proliferation of fibrous tissue as a result of a long continued inflammatory process. In this group, the changes have progressed beyond the stages of chronic catarrh and cholesterosis. There is proliferation of the connective tissue of the walls, the surface is contracted, the

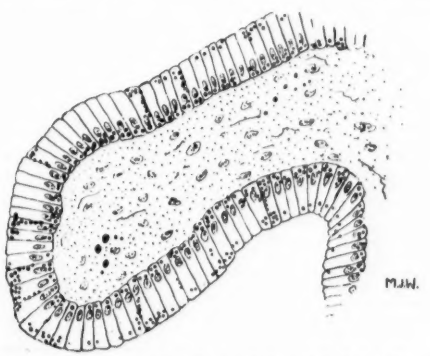

Fig. 3. Deposits of cholesterol in epithelial cells and stroma.

Fig. 4. Chronic cholecystitis; ridges of scar tissue may be seen.

walls are thickened with ridges of scar tissue, and there is loss of elasticity (Fig. 4). The mucosa is in part or wholly replaced by connective tissue, and there is lymphocytic infiltration.

There were 329 cases (7 per cent) of this type in the series; in 294 (89 per cent) of these, stones were present. The histories of 25 patients were studied. The average age was 43 years; there were three times as many women as men, the average duration of symptoms was 9 years, and the average weight was 144 pounds. There was nothing unusual or characteristic in the clinical histories except that the symptoms possibly were more severe and of longer duration than in the former types. About 30 per cent had a history of jaundice, and 10 per cent had chills and fever. Thirteen patients who had had gastric analyses had an average total gastric acidity of 50 and an average free hydrochloric acid value of 32. Achlorhydria was not present in this group.

Fig. 5. Hydrops of the gall bladder; stone impacted in cystic duct.

SUBACUTE AND ACUTE CHOLECYSTITIS

In subacute and acute cholecystitis there is active inflammatory reaction as a result of acute infection. Such gall bladders show injection and œdema of the walls with or without purulent changes, and on microscopic examination leucocytic infiltration is seen. The streptococcus, as shown by Rosenow, is the most common offending organism.

There were 319 specimens (6.9 per cent) of this type in the series, of which 308 (96 per cent) contained stones. Forty-six were considered to be acutely inflamed and 273 subacutely inflamed. About half of these gall bladders also showed evidence of marked chronic fibrous cholecystitis, as manifested by fibrosis and marked thickening of the walls. The histories of 25 patients were studied. The average age was 47 years. Chills and fever occurred in 10 per cent and attacks of gall bladder colic in 75 per cent. At the time of examination of these patients, the gall bladder was palpable in about a third of them. The temperature on admission varied from only

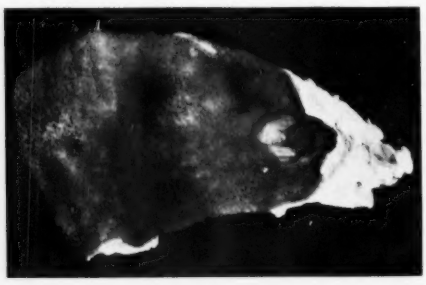

Fig. 6. Single papilloma in a chronic catarrhal cholecystitis.

Fig. 7. Numerous papillomata in chronic catarrhal cholecystitis.

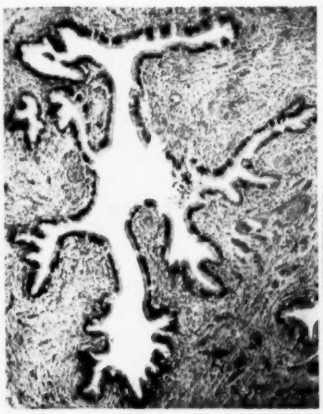

Fig. 8. Adenoma of the gall bladder; branching acini are shown.

Fig. 9. Carcinoma; acini filled with malignant cells in carcinoma simplex.

Fig. 10. Adenocarcinoma with mucoid production.

99.2 degrees to 100 degrees. The highest leucocyte count was 17,000; in only two other cases was it more than 10,000. Marked tenderness was elicited in practically all cases. The gastric content was not examined.

EMPYEMA OF THE GALL BLADDER

Empyema of the gall bladder is the result of an infection superimposed on obstruction of the cystic duct. The pathological picture is the same as that which exists in other organs in which pus forms, with thickened walls and diffuse inflammatory reaction.

There were 109 specimens (2.3 per cent) of this type in the series. Stones were present in 105 cases (96 per cent).

The histories of 25 patients were studied. The average age was 50 years, and the average weight was 150 pounds. Clinically these patients could be classified in two groups. In one group, comprising about 90 per cent, there was a long history of repeated colic and little, if any, suggestion of any purulent or inflammatory process. It might be expected that the chronic empyema type would fall in this group. In the smaller group there was a relatively short history of colic or soreness; the purulent process was evidently coincident with the first attack, namely acute empyema. As Moynihan (1928) stated: "The clinical conditions associated with empyema of the gall bladder vary greatly in severity and are in direct proportion to the intensity of the infection; in the more chronic forms the symptoms may be a little more acute than in hydrops, while in the more acute they are so grave that a fatal result may occur within a few days, all depending upon the severity of the infection." The temperature of the patients in this group at the time of examination varied from normal to 100 degrees; only 4 patients had a temperature of 100 degrees F. or slightly above. The average leucocyte count was 9,000 in each cubic millimeter of blood with 11,000 the upper limit. The gall bladder was palpable in six cases (24 per cent). The gastric content was not examined.

GANGRENE OF THE GALL BLADDER

Gangrene of the gall bladder may be produced by the toxins of the infective agent, or in some cases may be due to occlusion of the cystic artery from embolism, thrombosis, or the pressure of a stone. In this type, acute rupture may occur, either into the duodenum, the liver, or the general peritoneal cavity. The frequency with which the rupture is noted varies greatly in the various larger clinics and hospitals. At The Mayo Clinic, according to this series, it was noted in about one in every thousand cases in which cholecystectomy was performed.

In the series there were 60 cases (1.3 per cent) of gangrene, and stones were present in

Fig. 11. Squamous cell epithelioma of the gall bladder.

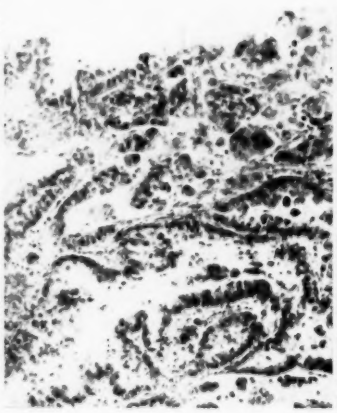

Fig. 12. Section of malignant papilloma.

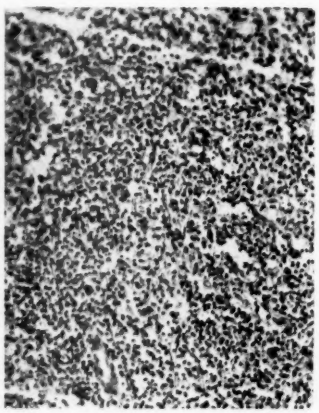

Fig. 13. Lymphosarcoma of the gall bladder.

58 (96 per cent). The histories of 25 patients were studied. The average age was 45 years; 50 per cent were men. The average weight was 168 pounds. The patients were all distinctly ill, much more so than any with the preceding types of cholecystic disease, and marked tenderness was noted in the right upper quadrant in all. About 20 per cent had chills and fever. The leucocyte count of 5 patients was more than 10,000 and of one it was 15,000. The temperatures, which varied from 98.6 degrees to 101 degrees, were above normal in only half of the cases. A mass was palpable in 30 per cent of the cases.

HYDROPS OF THE GALL BLADDER

Hydrops of the gall bladder (Fig. 5) is produced by obstruction of the cystic duct either from a stone becoming lodged in the duct, or, rarely, by inflammatory or other mechanical constriction at the neck of the gall bladder. The gall bladder occasionally becomes enormously distended and has been reported as being so large as to be mistaken for ovarian cyst. The incidence of hydrops reported by MacCarty in 1910 was 20 per cent, in 1919, 2.7 per cent; the difference is undoubtedly due to earlier removal of gall bladders in later years.

In the series there were 158 cases of hydrops (3.4 per cent); in 152 (96 per cent) stones were present. In the 6 remaining cases the hydrops might be considered secondary to inflammatory or other mechanical constriction. The histories of 50 of the patients were studied. The average age was 40 years, the average weight was 167 pounds, and 80 per cent gave a history of gall-stone colic. The patients did not appear so ill as those suffering from inflammatory or gangrenous cholecystic disease, and tenderness on palpation was much less. A mass was palpable in only 18 per cent of the cases. In 20 cases the gastric content was examined. The average total acids were 52 and the average free hydrochloric acid, 33; achlorhydria occurred in 2 cases.

PAPILLOMA OF THE GALL BLADDER

In these cases the cells cover finger-like processes or ridges of stroma. The papillomata usually occur singly (Fig. 6), but occasionally the entire surface of the gall bladder is literally studded with bunches of these finger-like processes (Fig. 7). The condition is primarily hypertrophy of one or more villi. They are light yellow, due to a deposit of cholesterol in the stroma. Microscopic sections in this series, stained with scarlet red, all showed a deposit of lipoid substance in the submucous stroma. The tumors vary in size from 1 to 4 millimeters. A single papilloma is frequently very small and, since it is extremely friable, is undoubtedly often overlooked.

In 417 of this series (9 per cent), one or more papillomata were observed; in 129 of these (30

SUMMARY OF DATA IN 4,575 GALL BLADDERS REMOVED AT OPERATION

	With stones		Without stones		Total	
	Cases	Per cent	Cases	Per cent	Cases	Per cent
Chronic catarrhal cholecystitis	1250	61	801	39	2051	44.8
Strawberry gall bladder or cholesterosis	552	50	542	50	1094	23.8
Chronic fibrous cholecystitis	294	89	35	11	329	7.1
Acute and subacute cholecystitis	308	96	11	4	319	6.9
Empyema of the gall bladder	105	96	4	4	109	2.3
Gangrene of the gall bladder	58	96	2	4	60	1.3
Hydrops of the gall bladder	152	96	6	4	158	3.4
Papilloma of the gall bladder	129	30	288	70	417	9.1
Adenoma of the gall bladder	6	25	18	75	24	0.5
Malignancy of the gall bladder	14	100			14	0.3

per cent) there were stones. The exact relationship between benign and malignant papilloma is problematic. Malignant changes occurring in papillomata have been reported, but evidence of malignant change was not present in any of these specimens. In a survey, however, of the various malignant conditions of the gall bladder, several papillary carcinomata were found.

The histories of 100 patients were studied. The average age was 44 years; the youngest patient was aged 21 years, and the oldest 68 years. The average weight was 140 pounds. The symptoms were not characteristic, but were simply those of cholecystitis in general, with or without stones. The gastric content was examined in 58 cases; the average total acids were 48, and the average free hydrochloric acid was 37. Achlorhydria occurred in 7 cases.

ADENOMA OF THE GALL BLADDER

In MacCarty's report in 1919, on about 5,000 gall bladders, there was only one adenoma. In fact, only a few adenomata are reported in the literature.

In this series of 4,575 gall bladders 24 adenomata were noted. The tumor was situated in the fundus of the gall bladder, an interesting fact inasmuch as normally glandular tissue is not present in the fundus, but its presence in some cases may explain in part the occurrence of adenocarcinoma in that situation. The average size of the adenomata was 1 centimeter; the smallest was 4 millimeters and the largest, 3 centimeters in diameter. Only six of the specimens contained calculi. On microscopic examination the adenomata were found to contain acini of columnar epithelium surrounded by connective tissue stroma, without evidence of malignant degeneration (Fig. 8). The histories of all of the patients in this group were studied. The average age was 46 years; the youngest patient was aged 28 years, and the oldest 62. The average weight was 133 pounds. The symptoms were those of cholecystic disease.

MALIGNANT LESIONS OF THE GALL BLADDER

The incidence of malignant lesions of the gall bladder is commonly given as ranging from 2 to 4 per cent. W. J. Mayo noted an incidence of malignancy of about 5 per cent. The incidence of malignancy in MacCarty's series (1919) was 0.5 per cent.

In this series there were 14 cases (0.3 per cent) of malignant lesions of the gall bladder. The types of malignancy of the gall bladder may be grouped as: (1) carcinoma simplex (Fig. 9); (2) adenocarcinoma (Fig. 10); (3) squamous cell epithelioma (Fig. 11); (4) papillary carcinoma (Fig. 12); and (5) sarcoma (Fig. 13). Carcinoma simplex and adenocarcinoma are the most common. These show branching acini of cylindrical cells, some of which surround a central lumen, others completely obliterating the lumen. Eleven of the fourteen lesions were either carcinoma simplex or adenocarcinoma. There were two cases of squamous cell epithelioma containing prickle cells, intercellular fibers, and horny cell nests. The condition is rare and is due to a process of metaplasia as a result of long continued irritation. Such change, when it does occur, however, is almost always associated with malignancy. There was one case of lymphosarcoma of the gall bladder. This condition is exceedingly rare; Goldstein (1921) was able to

find only 16 cases in the literature. Calculi were found to be a constant factor in association with malignancy of the gall bladder. In Deaver's experience stones occurred in 87 per cent of cases, and usually higher percentages are reported. All of the specimens in the group reported were associated with stones.

The histories of all of the patients in this group were studied, as well as those of about 50 other patients. The average age was 55 years. Usually there was a long history of repeated colic. Clinically, the cases could be grouped under three heads. In the first group, about 22 per cent, symptoms were present of an apparently harmless type of cholecystic disease, namely flatulence, intolerance to food, belching, and vague pain or tenderness, without loss of weight or appetite. In the second group, 70 per cent, there was a long history of repeated attacks of colic, followed by a history of from 1 to 3 months in which sudden change was characterized by continuous severe pain, anorexia, and progressive loss of weight. In the third group the histories were relatively short with reference to colic, 1 to 3 months, in which the malignant phase of anorexia, loss of appetite, loss of weight, and pain occurred coincidentally with the first symptoms of cholecystic disease. The patients in the two last groups lost on an average of 30 pounds in less than 6 months. In half of the cases a mass was palpated; in three there was jaundice. The blood picture was not associated with anæmia in any case. The average hæmoglobin was 73 per cent, and the hæmoglobin was not below 60 per cent in any case. The average total gastric acids were 39, and the average free hydrochloric acid was 31. In two cases achlorhydria was present.

SUMMARY

A simplified grouping of lesions of the gall bladder has been prepared in a series of 4,575 gall bladders, and the clinical manifestations in each group have been studied for comparison. There are no characteristic symptoms or clinical data to differentiate the apparently earlier types of cholecystic disease; in the later stages, however, certain characteristic data are present.

The clinical features of chronic catarrhal cholecystitis and strawberry gall bladder, or cholesterosis, were identical. The occurrence of stones in a fairly high percentage of gall bladders showing minimal pathological change further substantiates the theory that gall bladders may assume a normal appearance between attacks.

Chronic fibrous cholecystitis showed a higher incidence of gall-stone colic, jaundice, chills, and fever than the groups of chronic catarrhal cholecystitis and strawberry gall bladder, or cholesterosis. Stones were present in 89 per cent of the cases in this group.

Acute and subacute cholecystitis may occur without appreciable increase in temperature or leucocytosis. Gall stones occurred in 96 per cent of the cases.

The symptoms of empyema of the gall bladder vary greatly. The chronic form usually is not accompanied by any grave manifestations; the acute form not infrequently is fulminating in character. Stones occurred in 96 per cent of the cases.

Gangrene of the gall bladder is associated with marked clinical manifestations. There is marked tenderness, and the temperature and leucocyte count are higher than in the acute forms. Stones occurred in 96 per cent.

Hydrops of the gall bladder was associated with stone lodged in the cystic duct in 96 per cent of cases. A mass was palpable in 20 per cent. The patients were not nearly so ill as patients suffering from the acute empyema or gangrenous types of cholecystic disease.

Papillomata occur more frequently than the reported frequency. The single papilloma is friable and easily overlooked. The relation between papilloma and malignancy is problematic.

Adenomata always occur in the fundus. Normally glandular tissue is not present in the fundus of the gall bladder; this may, however, explain the frequent occurrence of adenocarcinoma in this situation.

The common types of malignant lesions of the gall bladder are: carcinoma simplex, adenocarcinoma, squamous cell epithelioma, papillary carcinoma, and sarcoma. Stones are a constant factor. Clinically malignant conditions of the gall bladder may be classified in two groups. In the first group the history is of

mild cholecystic disease; in the second group, of long duration of colic with a short terminal phase of loss of appetite, loss of weight, and constant pain; and in the third group, a short history of colic with a coincident malignant phase of loss of weight, anorexia, and pain.

BIBLIOGRAPHY

1. Boyd, William. Studies in gall-bladder pathology. Brit. J. Surg., 1923, x, 337–356.
2. Corkery, J. R. Chronic catarrhal cholecystitis with lipoid deposit. Ann. Surg., 1922, lxxvi, 736–738.
3. Deaver, J. B. Carcinoma of the gall bladder. Am. J. Surg., 1924, xxxviii, 105–107.
4. Goldstein, H. I. Primary sarcoma of the gall bladder. Am. J. Surg., 1921, xxxv, 351–352.
5. Judd, E. S. Clinical versus pathological cholecystitis. Proc. Interst. Post-Grad. M. Assembly N. Amer., 1925, 81–83.
6. MacCarty, W. C. The pathology of the gall bladder and some associated lesions. Ann. Surg., 1910, li, 651–669.
7. Idem. The frequency of the "strawberry" gall bladder. Ann. Surg., 1919, lxix, 131–134.
8. Mayo, C. H. Gall-bladder disease. New York M. J. 1916, ciii, 433–436.
9. Mayo, W. J. Malignant disease involving the gall bladder. Med. News, 1902, lxxxi, 1105–1107.
10. Mentzer, S. H. Cholesterosis of the gall bladder. Am. J. Path., 1925, i, 383–388.
11. Idem. A clinical and pathological study of cholecystitis and cholelithiasis. Surg., Gynec. & Obst., 1926, xlii, 782–793.
12. Moynihan, B. G. A. A disease of the gall bladder requiring cholecystectomy. Ann. Surg., 1909, i, 1265–1272.
13. Idem. A. The gall bladder and its infections. Brit. M. J., 1928, i, 1–6.
14. Rosenow, E. C. Bacteriology of cholecystitis and its production by injection of streptococci. J. Am. M. Ass., 1914, lxiii, 1835–1836.

EXPERIMENTAL PERITONITIS – II
THE EFFECT OF HYPERTONIC DEXTROSE SOLUTION UPON EXPERIMENTAL DIFFUSE PERITONITIS[1]

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IN a preliminary report of experimental work, presented two years ago, (1) we demonstrated that—

1. The transudate produced by the intraperitoneal injection of hypertonic dextrose solution enabled maintenance of the patency of rubber tube drains introduced within the peritoneum.

2. Adhesions between inflamed loops of bowel could be prevented by means of the mechanical isolation produced by the transudate and the absence of fibrin resulting from the great dilution of the normal serous exudate.

3. A transudate of considerable volume could be maintained in the peritoneum of the experimental animal without obvious harm.

According to Kuhn, who has written extensively on the use of intraperitoneal hypertonic dextrose solution, "sugar stops the toxin formation of all bacteria in the peritoneal cavity and replaces it by the fermentative herbiverous metabolism, which results in harmless acid products instead of injurious alkaline ones. It favors isolation, irrigation and fibrinolysis." Kuhn reports that with the use of dextrose, "cases that seemed doomed, have been saved." Reschke, in the most extensive research published in this subject, verified Kuhn's observation of delayed absorption from the peritoneum in the presence of dextrose. Narat produced what he considered a satisfactory lethal standard in 20 rabbits and reported recovery in 14 of 18 rabbits after intraperitoneal injection of 20 per cent dextrose solution. Clairemont, Haberer, and other investigators used glycerine collodein, camphor, zinc paste, and olive oil to lessen peritoneal absorption.

There are two mechanisms in wide-spread peritonitis, either one of which may produce death: obstruction through ileus and toxic absorption from the inflamed peritoneal surface and the contained exudate.

In the absence of ileus, we may assume that the toxæmia results from the absorption from the peritoneum of bacteria and their products, and that the resorption of the exudate itself plays a powerful, if not leading, rôle in the production of this toxæmia. In a like manner, it is logical to assume that any process that will (1) delay the rate of absorption and (2) remove the larger percentage of the toxic exudate, will be a factor in lowering the mortality rate. Physicians are familiar with conditions that slow absorption. In addition to the substances mentioned, cold, diminution of peristalsis, and fibrin also slow the rate of absorption. David called attention to the inhibitory influence of fibrin upon the absorption of diphtheria toxin and colon bacilli. It is conceivable that with the source of a given peritonitis under control, if it were possible to extend or slow by several days the resorption of a vast amount of toxic exudate, recovery might replace an otherwise fatal issue.

Exudation and absorption play the outstanding rôles in peritonitis. The entire mechanism of the peritoneum is concerned with the attempt to localize and limit the extent of serosa involved. Fibrin plays the major rôle in this mechanism. By its capacity for producing adhesions and encapsulation, fibrin limits or slows the spread of suppuration. By its close adhesion to inflamed surfaces, it delays absorption.

The diffusion of an inflammatory process in the peritoneum is dependent on or follows a deficiency in fibrin formation. The abundant, thin, seropurulent exudate of generalized peritonitis is familiar. In our experimental work we have noticed a striking, inverse, quantitative relationship between fibrin and virulence. Animals which died in 2 days or less showed no fibrin or at best scant traces. It is obvious, therefore, that any procedure which interferes with, or prevents fibrin formation, would be a dangerous one.

[1] Read before the Surgical Society, May 3, 1920. From the Laboratory of Surgical Research and the Department of Surgery, Northwestern University Medical School.

In our preliminary experimental work with the normal peritoneum we found it possible to exhaust or at least markedly diminish the absorptive capacity of the peritoneum. A single injection of 200 or 300 cubic centimeters of a 20 per cent dextrose solution that routinely produced a transudate approximately double that amount within the first 12 hours, almost always left the peritoneum fluid-free at the end of 24 hours. We discovered, however, that when such a transudate is maintained for 5 or 6 days, almost an equal amount of time was required for complete absorption of the transudate. A number of observers have discussed the apparent slowing in the absorption rate in cases of wide-spread peritonitis of several days' duration, as contrasted with the rate of absorption at the beginning of the disease. There can, of course, be little doubt that the rate of absorption must be materially delayed in the interval between the injection of a hypertonic solution and the establishment of an isotonic balance.

This then we considered our problem: To add to the exudate a transudate produced by hypertonic dextrose solution with the idea that: (1) absorption from the peritoneum will be slowed by the transudation and dilution of the exudate, and that, in addition, removal of larger amounts of this exudate than hitherto have been possible, could be accomplished by drainage. Since in widely disseminated peritonitis there is a fibrin failure, the addition of a factor capable still further of inhibiting fibrin did not seem to us to be detrimental. We did not engage the problem of peritoneal absorption—one that is as yet by no means settled—and we shall not take up the discussion of the mechanism of absorption of crystalloids, colloids, and particulate matter, or of the complex nature of the exudate itself.

The first phase of this experimental work was concerned with obtaining a satisfactory lethal standard. In the countless papers that have been published on experimental peritonitis, the majority of experiments have been conducted with bacterial cultures or pus from other sources. We sought a peritonitis that duplicated in its etiology and clinical course, as much as possible, the peritonitis of man, with the following characteristics:

1. The animal's own intestinal tract should be the source of the peritonitis because of discrepancies that would be introduced by the use of cultures.

2. The peritonitis should be diffuse to generalized. A sharp distinction must be made between a large, localized abscess and a generalized peritonitis.

3. The peritonitis must as nearly as possible be lethal, but not destroy the animal in 1 or 2 days, as it is utterly impossible to influence in any way an infection of such overwhelming virulence.

The procedure used was as follows: A segment of bowel of varying size was resected but left intact to its mesentery to insure its viability. Both ends were left open and henceforth this segment will be referred to as the "open loop." An end-to-end anastomosis was made around this loop and the opening in the mesentery closed. The omentum was wrapped about the suture line of the anastomosis and the bowel with the loop returned to the abdomen and a complete closure made. Autopsies were done as soon as possible after death and observations made on the following points: (1) the extent of the lesion; (2) the character and quantity of the exudate; (3) the bacteriology; and (4) the condition of the anastomosis.

EXPERIMENTAL WORK

Group I. In a series of 9 dogs a 6 inch open loop of the lower jejunum or upper ileum was used. There was one anæsthetic death in this group, leaving 8 for consideration. Four dogs (50 per cent) of this group died of generalized peritonitis, 3 of them in 3 days, 1 at 11 days. One died at 107 days of multiple abscesses, one in 95 days of pneumonia with a low-grade peritonitis, and one of pneumonia at 110 days. One died in 7 days of intestinal obstruction due to kinking.

Group II. In a series of 6 dogs an open loop of the lower jejunum 12 inches in length was used. There were 2 deaths from diffuse peritonitis; 1 at 1 day due to a leaky anastomosis; 1 at 7 days. The remaining dogs lived 34, 46, 76, and 90 days. Three died of pneumonia and 1 died of pneumonia and a peritoneal abscess.

Group III. In a series of 13 dogs, a 14 inch open loop of lower jejunum was used. There

was one anæsthetic death, twelve animals being left for consideration. There were 8 deaths from generalized peritonitis, of which one was due to leaky anastomosis. Excluding the latter, the average duration of the disease was 5 days. Three dogs were killed at 30, 50, and 61 days, respectively. In 2, the peritoneum was clean. In 1, there was a considerable quantity of mucilaginous material. One died at 81 days of multiple peritoneal abscesses.

Group IV. In a series of 8 dogs, the tip of the appendix was amputated and left open and the abdomen was closed. There were 3 deaths from generalized peritonitis; 2 in 1 day; 1 in 4 days. One died from a large localized abscess in 3 days. Two died of a local abscess in 10 and 66 days. At biopsy on the thirtieth day, one was found to have a localized abscess.

Group V. In a series of 13 dogs, a larger opening in the appendix was made than that in Group IV, in order to provide more rapid and wide-spread soiling. The animals were re-opened at the end of 22 to 26 hours and the opening in the appendix was closed by suture. There was one anæsthetic death. There were 5 deaths of generalized peritonitis; 2 in 1 day, 3 in 2 days. Biopsies made on 4 at 8, 10, 12, and 14 days disclosed localized abscesses. The biopsy on 1 at 100 days showed a mass of dense adhesions.

Group VI. In a series of 7 dogs the appendix was ligated with its mesentery, after the manner suggested by Costain. Two dogs died of generalized peritonitis at 2 days. One died on the sixth day of a large localized abscess. At biopsy, one on the eighth day showed a moderate-sized abscess. Biopsies on 3 others, on the fourteenth, eighteenth, and twenty-third days, showed the peritoneum to be clean.

Group VII. In a series of 7 dogs an open loop of colon 1.5 inches in length was used. There were 2 accidental deaths in this series; 1 from anæsthesia and 1 one from a leaky anastomosis. The remaining 5 died of a severe generalized peritonitis; 2 in 1 day, 1 at 2 days, 1 at 3, and 1 at 5 days.

Group VIII. In a series of 34 dogs, an open loop of mid-ileum 20 inches in length was used. There was 1 anæsthetic death and 2 deaths from leaky anastomosis. Of the remaining 31, there were 28 deaths from generalized peritonitis; 1 death from a large, localized abscess. One dog was used through error by one of the investigators for other work, and no autopsy was obtained. One animal survived, and a biopsy was done 100 days later. There was still a low-grade, diffuse peritonitis, with a fibrinopurulent exudate, and many diplococci were found in smears. The average duration of the disease was 4 days; 2 animals died in 1 day, 3 lived 6 days, 4 lived 7 days, 1 lived 9 days, and the remainder died on the second, third, fourth, and fifth days. This lesion, we decided, was the control that we were seeking.

DISCUSSION

The difficulties of producing a satisfactory lethal standard of generalized peritonitis are demonstrated in this series of 108 dogs. The peritoneum of the dog will tolerate an astounding degree of contamination, from intestinal contents, without producing death. We failed to get a mortality above 75 per cent, in cases in which a 14 inch loop of lower jejunum was used. The use of an open loop of colon, though a short one, produced so rapidly fatal a lesion as to make any form of experimental therapy impossible.

The greater frequency of local peritonitis following lesions of the appendix is worthy of mention. We must take issue with Costain, who stated that ligation of the appendix and meso-appendix invariably produced death from diffuse septic peritonitis in about 2 days. In a total of 28 animals, in which the appendix was used, only 10 developed generalized peritonitis and died. Four more died of local peritonitis, and 5, in which the lesion did not prove fatal, were found at biopsy to have local peritonitis. Lehman and Copher found this same discrepancy in Costain's controls.

The use of a 20 inch open loop of ileum gave a satisfactory lethal standard; the average duration of the disease being 4 days, and the mortality from generalized peritonitis 90.3 per cent. The pathological findings were quite uniform. They consisted of a peritonitis that involved practically the entire abdomen. The radiation was always quite obviously from the open loop, one end of which was almost invariably plugged by omentum. Encapsulated or

partially encapsulated abscesses were frequent in the vicinity of the open ends of the loop. The amount of fibrin in evidence was usually inversely proportional to the duration of the disease. In the most rapidly fatal form observed in animals dying in 1 and 2 days, fibrin was absent.

The bacteriology of these animals presented some points worthy of mention: The colon group was, of course, omnipresent, and the staphylococcus was frequently encountered. The streptococcus was absent, or present in small numbers, in animals that survived the mild, diffuse infections, or in those developing encapsulated lesions. In our rapidly fatal cases, the streptococcus was present in large numbers, or predominated. Two features characterized the latter cases; abundant, thin exudate and subserous petechial hæmorrhages. It is interesting to note that an animal harbored many staphylococci and a fibrinopurulent exudate for a period of over 3 months.

INTRAPERITONEAL DEXTROSE

In a series of 37 dogs, the usual 20 inch open loop of ileum was made and the abdomen closed. Twenty-four hours later, when a diffuse spreading peritonitis was assured, 20 per cent dextrose solution was injected into the peritoneum hypodermically. About 50 per cent of the maximum safe dose of dextrose was given at the first injection. It has been demonstrated that the normal animal will tolerate an intraperitoneal injection equivalent to approximately 1 per cent of its body weight in dextrose.

The animal was given repeated injections of approximately the same dosage at intervals of 12 hours. In the first 10 animals we did not give a hypodermoclysis of normal salt solution. These dogs obviously suffered from dehydration, a symptom that was not noticed in the control animals which were given no dextrose. It is far more difficult to control dehydration in a dog than in man. In the next 17 animals, normal salt solution was injected subcutaneously at the time that the dextrose solution was given intraperitoneally in varying amounts up to twice the volume of the dextrose solution given. The average length of life after operation of the first 10 animals was 1.75 days. Of the next 17, it was 2.2 days. One animal survived after injections over a period of 5 days. Biopsy on the seventy-seventh day revealed the abdomen partially filled with a mucilaginous material, relatively free from adhesions, excepting those between the omentum and the open loop. Smear showed bacilli and diplococci present. The amount of exudate present at autopsy in this series of 37 animals was 50 per cent to 100 per cent greater than in the control animals and showed a thinning in consistency that would be expected from the increase in peritoneal secretion produced by the dextrose solution. In these animals there was a definite diminution in the amount of encapsulation about the open loop.

In the next 10 animals, following the usual loop resection, a soft rubber tube drain was sutured into the peritoneum and dextrose injected hypodermically through the right hypochondrium and through the tube, which was in the lower abdomen. We attempted to maintain the patency of the tube and communication between the latter and the free peritoneal cavity, injections being given, as in the previous series, every 12 hours. One animal survived and is still alive. The remaining 9 died of generalized peritonitis; 2 in 1 day, 4 in 2 days, 1 in 3 days, and 2 in 4 days—an average duration of $2\frac{1}{3}$ days. Two of the animals drained in such large amounts as to indicate that the drain was not encapsulated. In the remaining 7, encapsulation occurred in spite of a copious exudate and wide distribution of the dextrose solution.

The dextrose solution did not effect the healing of the suture line of the end-to-end anastomosis. We have previously had occasion to note that such a suture line will remain intact and heal normally in the presence of a fibrin-free transudate. It would seem, therefore, that fibrin is not essential to the safety of a peritoneal suture line nor its future healing, but at autopsy it was apparent in this series of animals that a more rapid spread of the peritonitis was due to the lavage of the transudate, and this resulted in death in an average of 2 days instead of in 4, as occurred in the controls.

COMMENT

Dextrose solution injected into the abdomen in the presence of a diffuse peritonitis during the period of spread, failed not only favorably to influence the mortality, but shortened the duration of life by 50 per cent. This action is probably due to a speeding up of the diffusion of the infection over a wide area of the peritoneal surface. In his experimental study one year ago, David suggested this point. The absorptive power of the peritoneum remained good through the second and third days of the disease, as was shown by the disappearance of the sugar from the peritoneal exudate at autopsy, but in those animals surviving 4 or 5 days, the sugar test was positive, ranging from 2 per cent to 4 per cent. The temporary delay in absorption, which we should expect to occur during the period when an osmotic balance between the hypertonic peritoneal exudate and the blood serum was occurring, was not sufficient to lower the mortality or even ameliorate the course of the disease. We may assume by analogy that the above facts would obtain in human cases; namely, that the temporary slowing of the absorptive capacity of the peritoneum caused by hypertonic dextrose solution would not modify the course of the disease.

The capacity of the peritoneum for encapsulation which is present in the normal membrane and in moderate grades of infection is absent, or markedly diminished, in the presence of virulent infection. Such a condition is, as we have stated, a fibrin failure, the result of the excessive dilution, produced by the abundant exudate that characterizes such rapidly spreading infections. There is, however, a striking paradox in the latter situation. In such a peritonitis, with no fibrin in evidence, one would assume the capacity of the peritoneum, for encapsulation of a rubber drain would be no greater than its capacity of encapsulation of the source of infection. Yet the omentum will more effectively plug a benign rubber drain than a bowel perforation or the open end of a contaminating loop, and the vicinity of the drain will quite likely show the only evidence of fibrin in the abdomen. This should re-emphasize the futility of any type of drainage in peritonitis.

This brings us to a consideration of a possible relationship between the exudate and paralytic ileus. In the few cases in man in which we have opened the abdomen because of paralytic ileus, we have been impressed by the scantiness, or even absence, of a fluid exudate. The dog does not die of paralytic ileus; we have not observed a single instance of ileus in more than 200 animals with peritonitis. But the dog's peritoneum produces an exudate proportionately greater in quantity than does the peritoneum in the human. In the abundant exudate present in practically every fatal case, the intestinal tract is literally bathed in exudate. It seemed to us that the mechanical isolation of the contiguous loops of bowel caused by the very abundance of the exudate is the prime factor in preventing ileus. In such cases we believe that the exudate acts as a mechanical isolator of the intestinal loops.

It is known that the intra-peritoneal introduction of hypertonic solutions stimulates peristalsis. Hypertonic dextrose solution will produce such a mechanical isolation of distended loops very satisfactorily. It is still to be determined whether or not in such a procedure we have a safe and satisfactory means of relieving intractable ileus with low-grade, but diffuse, peritoneal infection.

In dealing with human peritonitis, we frequently encounter a diagnostic problem that can be settled only at autopsy; this is the extent of involvement of the peritoneum. The clinical signs by which we diagnose the disease will usually not serve to define its extent. Experimentally we have demonstrated to our own satisfaction that the animal with a rather wide-spread, diffuse peritonitis may live. The animal with complete, or nearly complete, involvement nearly always dies. It takes, however, a surprisingly long time to involve all of the peritoneum even in the presence of rapid, wide-spread soiling where localization or encapsulation does not occur and where even traces of fibrin are not observed. In such animals, who uniformly died at the end of 4 days of a generalized peritonitis, at the end of 24 hours there was present a diffuse lesion that did not involve in excess of one-third of the peritoneal surface. Even the most mobile of the viscera, the small bowel, is only partially

involved at this time, yet in some instances it took 7 or 8 days for the lesion to spread to the diaphragm and to the pelvis.

How then can we reconcile the results of this experimental work with the several reports that describe in glowing terms the results of hypertonic dextrose solution in severe human peritonitis?

It will probably never be possible to describe a lethal standard in the human. It is notoriously difficult to judge the virulence of the disease or its extent by the clinical picture except in moribund cases without wide-spread exploration. It is possible to introduce dextrose, as has been done, through a small incision into a part of the peritoneum not actively involved in a septic process. If the primary focus is already encapsulated, the fibrinous exudate responsible for this encapsulation will be unaffected by the transudate in the free peritoneum. Kuhn refers to the fibrinolytic action of dextrose. We have not observed any marked solvent action of dextrose on fibrin, such as is found in Dakin's solution. It would be utterly illogical to attempt anything at all with hypertonic dextrose solution in the peritoneum, if it had such a fibrinolytic action, because it would promptly convert a local into a spreading process.

CONCLUSIONS

1. Rapidity of absorption in the largest measure governs the prognosis of acute, diffuse peritonitis.

2. Fibrin is the most important factor in controlling the rate of absorption.

3. Fibrin is diminished or absent in the more virulent cases because of dilution of the exudate.

4. The streptococcus is most commonly identified with this abundant exudate and the accompanying virulent course of the disease.

5. The addition to such an inflammatory exudate of a transudate produced by the intraperitoneal injection of hypertonic dextrose solution produces a more rapid spread of the infection and insures the lethal outcome.

6. It seems probable that an abundance of thin exudate serves to prevent ileus by mechanical isolation of intestinal loops.

7. The results of this experimental study do not agree with the published reports of the similar treatment of peritonitis in man.

BIBLIOGRAPHY

1. Buchbinder, J. R. Prevention of peritoneal adhesions and encapsulation: preliminary report of an experimental study of peritoneal reaction to hypertonic dextrose solution. Surg., Gynec. & Obst., 1927, xlv, 769–774.
2. Costain, W. A. Lymphaticostomy in peritonitis. Surg., Gynec. & Obst., 1923, xxxvi, 365–367.
   Idem. Brit. M. J., 1924, ii, 857–858.
3. David, V. C., and Sparks, J. L. The peritoneum as related to peritonitis. Ann. Surg., 1928, lxxxviii, 672–677.
   Idem. Peritonitis; experimental study. Surg., Gynec. & Obst., 1927, xlv, 287–293.
4. Kuhn, F. Die biologische Behandlung der Peritonitis. Muenchen. med. Wchnschr., 1911, lviii, 1998–2002.
   Idem. Die Zuker Behandlung der Bauchfellentzuendung. Arch. f. klin. Chir., 1911, xcvi, 759–896.
   Idem. Die biologische Behandlung der diffusen eitrigene Bauchfellentzuendung. Zentralbl. f. Chir., 1926, liii, 1108–1112.
5. Lehman, E. C., and Copher, G. H. Lymphaticostomy; experimental and clinical study of thoracic duct drainage in peritonitis. Arch. Surg., 1925, x, 742–750.
6. Narat, J. K. Experimental study upon the use of intra-abdominal injections of hypertonic glucose solution in the treatment of peritonitis. Ann. Surg., 1923, lxxviii, 357–363.
7. Reschke, K. Versuche ueber die Beeinflussung der peritoneal Resorption durch hypertonische Losungen zwecks Anwendung. Arch. f. klin. Chir., 1921, cxvi, 466–521.

THE ABSORPTION OF GLUCOSE FROM THE COLON

A Preliminary Study of the Glucose Enema[1]

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THERE exist many missing links in our knowledge of the absorptive power of the large intestine. That certain substances enter the circulation of the human body when placed into the rectum is a commonly known fact. As Sollmann tells us, ether is readily taken up by the lower bowel and complete general anæsthesia is produced; this is a well known clinical phenomenon. Bromides are likewise absorbed when placed into the colon, and the bromide ion can be detected experimentally in the urine, the sweat, the tears, and other body secretions. It is less well known, however, how useful the sugar enema and other so-called nutritive rectal administrations are. No modern textbook of physiology, dietetics, or nutrition lacks a discussion on rectal feeding. Yet, a careful perusal of these writings will impress the reader with their delicious vagueness and will leave with him the suspicion that a great many statements are founded on insufficient evidence, not to say speculation.

The medical and biological journals of the last 15 years contain a voluminous mass of information on the therapeutic administration of glucose. However, when one sifts out those articles dealing with oral, subcutaneous, and intravenous methods, there are left but scant experimental data on the colon as the portal of entrance of glucose into the body. Furthermore, it is still a matter of considerable controversy whether the rectum absorbs efficiently any nutritive substance.

REVIEW OF THE LITERATURE

From Tallerman we learn that 180 cubic centimeters of a 33 per cent glucose solution injected slowly into the human rectum produce a slight and slow rise in the peripheral blood sugar level. Levi injected 500 cubic centimeters of glucose solution of strengths varying from 10 per cent to 16 per cent into the large bowel of normal fasting men, diabetic patients, and patients operated upon and examined the peripheral blood for its sugar content. He concluded that the large bowel varied in different individuals as to its absorptive power, but that at best only small quantities of glucose were absorbed. Large amounts of 40 per cent glucose solution were introduced by Varela and Rubino into the rectal ampulla of patients and the peripheral blood and the urine tested for sugar. They claim that minute amounts are absorbed shortly following introduction, and that soon thereafter the colon becomes irritated and expels the enema. Franke and Wagner gave highly concentrated glucose enemas to dogs, and they state that they observed very little effect on the peripheral blood sugar level. Reach investigated the respiratory quotient after rectal injection of glucose and found that there was some absorption, slow and slight though it was. Hari and Halasz, working on dogs, placed a ligature around the ileocæcal valve and obtained a slight change in the respiratory quotient. Ornstein performed an experiment which is wide open to criticism. He introduced large quantities of sugar and starch mixed with proteins and fats into the rectum of dogs, and from analysis of the urine and washed out fæces, 6 to 8 hours later, concluded that all the sugar and some of the starch were absorbed. McLester, Friedenwald and Ruraeh, and Hughson lead us to believe, without experimental basis, that glucose given by rectum is a valuable nutritive procedure. A different view is held by Nelson, who states that glucose placed into the large bowel is irritating and not often retained.

COMMENT

The evidence gained from these various authors leaves us in a quandary as to the value of the glucose enema. Conflicting as these reports are, still certain facts emerge which are worthy of consideration:

First, the solutions used by these workers are, without exception, to a more or less

[1] This study was made in the Department of Physiology, Northwestern University Medical School, and we were greatly aided by valuable suggestions and friendly criticisms from Dr. A. C. Ivy, head of that Department. Read before the Chicago Surgical Society, April 5, 1929.

degree hypertonic with the blood and tissue fluids. Theoretically, a 4.9 per cent glucose solution has the same osmotic tension as a 0.9 per cent sodium chloride solution. Kosaki found that a strength of 7 per cent was the most satisfactory, while strengths of 20 per cent and over caused destruction of the erythrocytes. Goldschmidt states that any solution of sufficient degree of hypertonicity will, when placed into the intestine, draw fluids into the gut. That this not only hinders absorption, but may actually become a danger to life is brought out by Hausmann who introduced a 50 per cent solution of saccharose into the peritoneal cavity of rats, which promptly died from toxicity caused by the withdrawal of large quantities of fluids from the tissues into the peritoneal cavity. We believe, therefore, that a 4.5 or 5 per cent solution of glucose is the optimum strength for all therapeutic administration, and that the high concentrations used by the various writers mentioned above are far too high to give conclusive results.

The *second* noteworthy fact in the experimental work cited above is that blood sugar estimations in all instances were made on the peripheral blood only. Starling tells us that the path of all absorption from the entire intestinal tract leads through the liver by way of the portal system. It is evident, therefore, that glucose absorbed from the intestine is submitted to the metabolic activity of the liver cells before it reaches the general circulation, and that a sugar determination made on the peripheral blood is not necessarily an index of the absorptive power of the intestine. In our work we have, therefore, made estimations on the portal blood, where rises in the blood sugar values represent actual absorption of the sugar solutions within the bowel, unaffected by the activity of the liver.

The *third* outstanding fact is that no author, to our knowledge, has taken cognizance of the fermentative possibilities of the colon, which is the normal reservoir for a huge number and variety of bacteria, many of which have specific actions on specific sugars. This phase of the problem we have not yet taken up in our work, except that we have been careful to forestall any appreciable decomposition of the sugar by bacteria by leaving the solutions in the bowel not longer than 1 hour and by titrating them immediately after removal.

EXPERIMENTAL WORK

In our experimental work we used a series of 15 dogs. These animals were picked at random, and they weighed from 20 to 35 pounds each. Food was withheld for about 16 to 18 hours. All the work was done under barbital hypnosis, 0.2 grams of sodium barbital per kilogram of body weight being given by stomach tube. One-half hour later a small amount of ether was given to induce rapid anæsthesia, after which the dog remained under light hypnosis throughout the experiment. The abdomen was opened and the entire colon isolated by ligatures around the ileocæcal valve and the rectosigmoid junction. A loop of the distal ileum was likewise isolated by a ligature. An effort was made to have the loop of small bowel 50 per cent longer than that of the large bowel, which, roughly speaking, gives approximately equal absorbing surfaces in both. With all possible gentleness we then inserted glass cannulas into the four ends of the isolated loops and washed the contents out of the bowels in a proximo-distal direction with normal salt solution warmed to body temperature. After the solution returned perfectly clear and unhindered the bowel loops were completely emptied by tilting the table up into such a position that the exit cannulas were at the lowermost points of the loops and by gently blowing warm air through them. Then an accurately measured quantity of 5 per cent glucose solution at body temperature was placed into each loop and the cannulas closed. This amount we varied from 50 to 75 cubic centimeters according to the size of the dog, so that the bowels were in no instance overdistended by the contained solution. All protruding intestines were then replaced into the abdomen, the incised edges of the parietes were brought together and closed with a hæmostat and covered with a warm towel. Exactly one-half hour later the residual solutions were removed and the loops washed out with a definite quantity of warm saline solu-

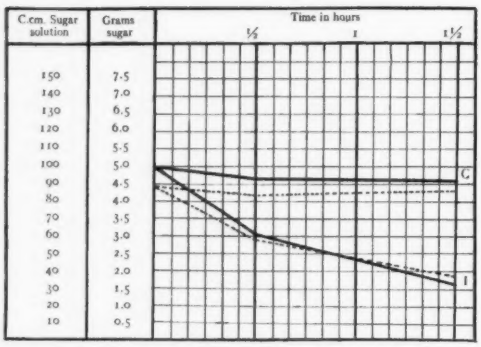

Chart 1. Dog E, male, 21 pounds. Glucose introduced into ileum, 100 c.cm., 4.42 gm.; colon, 100 c.cm., 4.42 gm. Glucose recovered from ileum, 31 c.cm., 1.81 gm.; colon, 92 c.cm., 4.36 gm. Glucose lost from ileum, 69 c.cm., 2.61 gm.; colon, 8 c.cm., 0.06 gm. Percentage recovered from ileum, 31 per cent c.cm., 41 per cent gm.; colon, 92 per cent c.cm., 98.7 per cent gm. Percentage lost from ileum, 69 per cent c.cm., 59 per cent gm.; colon, 8 per cent c.cm., 1.3 per cent gm. Solid line indicates glucose solution, broken line glucose; *C*, colon; *I*, ileum.

tion. Next, the same volumes as before of 5 per cent glucose solution were again introduced into the bowel loops and allowed to remain 1 hour, after which the residues were recovered as before and the loops washed out to recover all glucose clinging to the mucosal folds or the walls of the funnel and cannulas. All the residual solutions recovered in this way were accurately measured to detect loss of water and were assayed by Benedict's quantitative method to determine loss of glucose. The saline washings were likewise measured and titrated in order to recover all possible glucose from the bowel loops. The total amount of solution introduced and recovered in cubic centimeters and the total quantity of glucose introduced and recovered in grams, were then calculated. In the process of each experiment the solutions were handled eight times, and, consequently, experimental errors in the form of loss of solution and of the contained sugar were unavoidable. After a considerable number of experiments we determined this error to be approximately from 1 to 2 cubic centimeters of solution and from 50 to 100 milligrams of sugar.

In all of these experiments we found a marked constancy in the action of the colon, in contrast to a marked variation in the activity of the ileum. The amounts of sugar lost in the colon were so small as to be accountable for on the basis of experimental error, while from the ileum relatively large quantities were absorbed. In several dogs there was a slight absorption of fluid by the colon, thus leaving the remaining glucose solution slightly more concentrated than when introduced. From the ileum, in every instance, there was absorbed a considerable amount of glucose. Chart 1 illustrates graphically the results in a typical dog. The maximum amount of glucose solution which we were able to recover from the ileum in 1½ hours was 82 per cent of the amount introduced; the minimum was 31 per cent. From the colon the corresponding figures are 96.6 per cent and 87 per cent, respectively. The maximum percentage of grams of glucose recovered from the ileum was 76.8 per cent; the minimum was 41 per cent. From the colon we recovered as little as 97.4 per cent and as much as 99.6 per cent of the original glucose introduced.

Summarizing these figures briefly in another way, we have a loss of glucose solution in the ileum varying from 18 per cent to 69 per cent; in the colon from 3.4 per cent to 13 per cent. Of the glucose introduced in terms of grams there was a loss in the ileum varying from 23.2 per cent to 59 per cent; in the colon from 0.4 per cent to 2.6 per cent.

Next we attacked the problem from the other side of the absorbing surface. In this group of experiments the dog was prepared in the same way, the abdomen opened, and the loops of ileum and colon isolated as before. Samples of blood were then taken from the large veins draining these loops. Then 5 per cent glucose solution was introduced as before. At intervals of 20 and 40 minutes blood samples were again withdrawn from the mesenteric veins of the isolated bowel loops. The sugar in the blood thus withdrawn was then determined by the colorimetric method of Folin and Wu.

The results were remarkably definite. In no case was there a rise in the blood sugar level of the blood from the colon loop; and in all instances there appeared a marked rise in

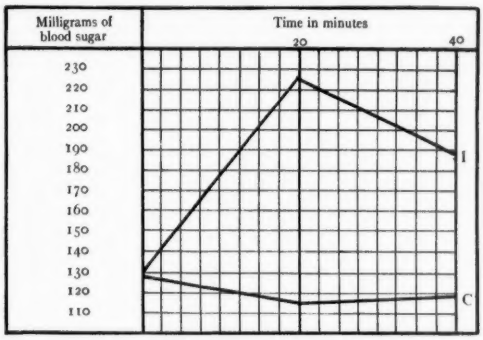

Chart 2. Dog N, male, 33 pounds. Change in blood sugar level following glucose introduction: *I*, Ileal vein, 130 mgm., 226 mgm., 186 mgm. *C*, Colic vein, 128 mgm., 115 mgm., 117 mgm.

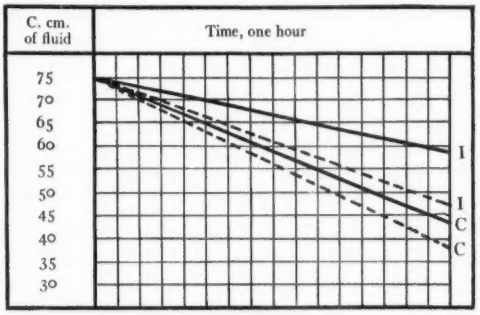

Chart 3. Dog O, female, 34 pounds. Fluids introduced into ileum, 75 c.cm.; colon, 75 c.cm. Tap water recovered from ileum, 59 c.cm., 78.6 per cent; colon, 43 c.cm., 57.3 per cent. Tap water absorbed from ileum, 16 c.cm., 21.3 per cent; colon, 32 c.cm., 42.6 per cent. Normal salt solution recovered from ileum, 47 c.cm., 62.6 per cent; colon, 39 c.cm., 52 per cent. 0.9 per cent sodium chloride absorbed from ileum, 28c. cm., 37.3 per cent; colon, 36 c.cm., 48 per cent. Solid lines, tap water; broken lines, saline; *C*, colon; *I*, ileum.

the blood from the loop of ileum. Chart 2 is typical of this type of experiment. It shows the blood sugar values immediately preceding and 20 and 40 minutes following the introduction of a 5 per cent glucose solution into the bowel loops.

The third type of experiment concerns the absorption of tap water and of physiological salt solution from the same bowel loops used in the previous work. The technique employed was exactly as before. Of the tap water placed into the loops we recovered in one hour from the ileum 78.6 per cent; from the colon 57.3 per cent. Of the physiological salt solution we recovered in one hour from the ileum 62.6 per cent; from the colon 52 per cent. This shows definitely that the colon has the power to absorb these substances and does it better than the ileum (Chart 3).

SUMMARY

In summarizing our work briefly it is probably safe to say that there is no appreciable absorption of glucose in 5 per cent aqueous solution from the colon, while from the ileum considerable absorption takes place. Tap water and normal salt solution are absorbed rapidly by both ileum and colon.

DISCUSSION

In discussing our results in the light of the known physiological functions of the colon it becomes apparent that when a glucose solution is placed into the rectum one or more of the following things take place: (1) it stays *in situ* unaltered, indefinitely, (2) it is expelled, (3) it changes in character by bacterial or other action, (4) it is absorbed, or (5) it passes into the small bowel.

In the first three instances the rectal administration can probably be best described as useless, if not indeed harmful. In using high concentrations of glucose solutions Tallerman, Levi, Franke, et al., Ornstein, and Varela, et al. probably introduced an irritant to the rectal mucous membrane, following which expulsion is the normal sequence. This is the view held by Nelson. It is also possible that, if not already irritating, a glucose solution can in a short time become so by the products of bacterial decomposition, chief among which are gas and lactic acid. We believe that glucose is not appreciably absorbed from the colon, but that on rectal administration of any considerable quantity of solution the ileocæcal valve becomes incompetent, and the glucose is regurgitated into the distal ileum, where absorption promptly takes place. Such regurgitation is very probably not a normal procedure in the colon. Cannon gave nutrient enemas to cats under the fluoroscope and found that a small enema will lie quietly in the descending colon,

but a larger one sets up antiperistaltic waves which force the fluid into the ileum. Gruetzner claims that substances which he introduced into the rectum were recovered in the stomach, being driven upward by antiperistalsis. Lurje made extensive studies on the actions of the colon and states that movements of tonic as well as antiperistaltic nature may occur in the proximal part of the colon. Case holds that the ileocæcal valve becomes insufficient when there is obstruction or back pressure in the large bowel. Nagel also states that substances introduced rectally are not absorbed there but enter the distal ileum. Opposed to this view is Goldschmidt when he claims that absorption from the entire length of the intestinal tract involves a similar mechanism, and that differences of absorption at various points are quantitative differences rather than qualitative. He believes further that the presence of sodium chloride favors the absorption of glucose by acting as a chemical excitant. The study of the absorption of mixtures of sodium chloride and glucose solutions in various proportions is the next phase in our problem to be reported on.

We are aware of the differences existing between the large intestine of the dog and that of man. Such differences are primarily anatomical and are less important functionally and physiologically. Yet they must be kept in mind in the application of experimental results to clinical phenomena.

CONCLUSION

In view of all the evidence brought forward the uptake of rectally administered glucose probably depends on the passage of the enema into the ileum through an incompetent ileocæcal valve. This very likely is the basis of success in the treatment of hyperemesis gravidarum by rectal glucose administration. The normally competent valve becomes insufficient on irritation or on sufficient pressure from below. Such a condition is not physiologically normal, and at best only small amounts of glucose can be forced into the blood stream in this way.

We conclude that a 5 per cent glucose enema is of little or no nutritional value.

BIBLIOGRAPHY

1. Cannon, W. B. The movements of the intestine studied by means of the roentgen ray. Am. J. Physiol., 1902, vi, 251.
2. Case, J. T. The X-ray investigation of the colon. Arch. Roentg. Ray, 1915, xix, 375.
3. Franke, W., and Wagner, R. J. Studies on the fermentable blood sugar after parenteral and rectal administration of glucose. J. Met. Research, 1924, vi, 375.
4. Friedenwald, J., and Ruraeh, J. Diet in Health and Disease. Philadelphia: W. B. Saunders and Company, 1919.
5. Goldschmidt, S. On the mechanism of absorption from the intestine. Physiol. Rev., 1921, i, 421.
6. Gruetzner, P. Zur Physiologie der Darmbewegung. Deutsche med. Wchnschr., 1894, xx, 897.
7. Hari, Paul, and Halasz, A. Ueber die Resorption des rektal eingefuehrten Traubenzuckers. Biochem. Ztschr., 1918, lxxxviii, 337.
8. Hausmann, W. Zur Kenntnis der toxischen Wirkung konzentrierter Zuckerloesungen. Wien. klin. Wchnschr., 1925, xxxviii, 332.
9. Hughson, Walter. Dean Lewis' Practice of Surgery, vol. i. Hagerstown, Maryland: W. F. Prior Company, 1928.
10. Kosaki, M. A study of immune hemolysin. J. Immunol., 1918, iii, 109.
11. Levi, David. A note on the glucose enema and its value in postoperative treatment. Brit. J. Surg., 1927, xv, 282.
12. Lurje, H. S. Untersuchungen ueber die motorische Funktion des Dickdarmes. II. Zur Frage ueber die Antiperistaltik des Dickdarmes. Arch. f. d. ges. Physiol., 1925, ccviii, 249.
13. Idem. III. Die tonische kontraktion der Dickdarmabschnitte als selbstaendige Form der Kontraktionen. Arch. f. d. ges. Physiol., 1925, ccviii, 255.
14. McLester, J. S. Nutrition and Diet in Health and Disease. Philadelphia: W. B. Saunders and Company, 1927.
15. Nagel, W. Handbuch der Physiologie des Menschen. vol. ii. Braunschweig: F. Vieweg & Sohn, 1907.
16. Nelson, Garnett. The use of glucose in medicine and surgery. South. M. & S., 1926, lxxxviii, 163.
17. Ornstein, L. Stoffwechselversuche mit rektaler Ernaehrung. Biochem. Ztschr., 1918, lxxxvii, 217.
18. Reach, Felix. Ueber Resorption von Kohlehydraten von der Schleimhaut des Rektums. Arch. f. exper. Path. u. Pharmakol., 1902, xlvii, 231.
19. Sollmann, T. A Manual of Pharmacology. 2d ed. Philadelphia: W. B. Saunders and Company, 1922.
20. Starling, E. H. Die Resorption vom Verdauungskanal aus. Handbuch der Biochemie des Menschen und der Tiere. C. Oppenheimer, Vol. III, Part II. Jena: Gustav Fischer, 1909.
21. Tallerman, K. H. On the rectal absorption of glucose. Quart. J. Med., 1926–27, xiii, 356.
22. Varela and Rubino. Rektale Dextrosezufuhr und Blutzucker. Med. Klin., 1922, xviii, 831.

SOME UTERINE ANOMALIES DUE TO VARIATIONS IN THE FUSION OF THE MUELLERIAN DUCTS

A Partial Review of the Literature with a Report of Nineteen Cases Seen at the Boston Lying-in Hospital and the Free Hospital for Women, Brookline, Massachusetts

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THE occurrence of double uterus is probably more frequent than is generally suspected. Since it is seldom recognized in general practice, its frequency, importance, and interest are underestimated. Falls believes that 1 per cent of all uteri are bicornuate. Another writer estimates that 14 per cent of all uterine anomalies are of the bicornuate type. This figure is probably too conservative judging by the cases reported in the past 30 years. Disregarding figures, however, duplication of the uterus is one of the last thoughts to enter the mind of the obstetrician or gynecologist when parturition is unaccountably protracted or uterine dysfunction remains undiagnosed.

Authorities agree that the distal ends of the muellerian ducts, being in apposition in the genital cord and flanked by the mesonephric ducts which exert pressure in a medial direction, fuse at the 20 to 30 millimeter stage of the embryo and result in the unpaired anlage of the uterus and vagina. The remaining parts of the muellerian ducts lying cephalad to the genital core persist as pairs and become the uterine tubes. During the twelfth to the fourteenth week of embryonic life the formerly approximated medial walls disappear and fusion is completed. Canalization takes place from below upward thus forming the potential cavities of the vagina and uterus, the process being completed between the twentieth and twenty-fourth weeks. This substantiates the view of Burrage, who declares that the presence of two vaginal canals is a definite indication of a double uterus. Byford, however, and several others hold that a septum may occur in a vagina or cervix only, the uterus being normally developed. Since cases with septate vagina and normal uterus are known to occur, it is likely that canalization may begin in any portion of the genital core.

The following is Reed's table giving the nomenclature and explaining female genital anomalies as they occur in the stages of development:

REED'S TABLE

Embryonic

1. (a) Absence of tubes, uterus, and vagina. This is very rare. (b) One-horned uterus with absence of the other horn (uterus unicornis sine ullo rudimento cornu alterius).

2. (a) Externally double uterus (uterus duplex sine didelphys, uterus bicornis). (b) Solid or partly excavated uterus (uterus solidus, uterus rudimentarius, uterus partim excavatus). (c) Combination of a and b (uterus duplex solidus, uterus bicornis rudimentarius). (d) One-horned uterus with the other horn solid or partly excavated (uterus unicornis cum rudimento cornu alterius).

3. Uterus divided internally more or less completely with or without external signs or duplicity (uterus septus subseptus, uterus bicornis septus).

Fetal

4. Uterus with flat fundus with or without complete or partial duplicity (uterus planifundalis septus, subseptus, simplex).

5. Uterus with fetal character (small body, large cervix).

Post-natal

6. Uterus with infantile character (uterus infantilis).

This table does not entirely cover uterine anomalies, as pointed out by Ballantyne who presented as an example the trifid uterus or uterus accessorius which is particularly difficult of embryonic explanation. Duplication of the muellerian duct on one side during the germinal period was given as a supposition.

Uterus didelphys, or completely double uterus internally and externally, is also missing in the above table.

Theories as to the cause of uterine duplication are varied, but the most acceptable is that described by Ancel and Villemin. Having found a case with a vesicorectal fold, they looked up 24 other cases with this fold and discovered that it occurred only in females and was accompanied in every instance by uterine duplication. They stated that the vesicorectal fold is vascular in origin, the segment from the upper rectum to the bladder being produced by a branch of the superior hæmorrhoidal artery and the remaining portion, which extends to the sigmoid, being formed by a collateral branch of that vessel.

Eden and Lockyer referred to the vesicorectal fold which they considered was either of allantoidal origin, being caused by fetal peritonitis or a relic of the terminal intestinal mesentery. Numerous other writers conceded that the vesicorectal fold was an etiological factor in uterine duplicity, but some, e.g., Nagel, deemed it the result.

Another theory is that the ducts of Mueller are held apart by abnormally short round ligaments. Piquand discussed the whole question of causation of length. Newton referred to Pick, who found tumors present in 30 cases of uterus duplex, and claimed that the presence of tumors had an etiological bearing. Felix and Rosenstein (31) stated that formative disturbances of the intestinal tract and developmental errors of the ventral abdominal wall were etiological factors. Von Franque substantiates this view with a report of four cases. Jacobs reported a case of bicornuate uterus in which there was a congenital absence of the appendix (agenesis). Like many other pathological conditions uterine anomalies are probably due to a combination of causes. Moench very well said that phylogenetic youth and intricacy of structure inherently cause vulnerability.

The literature deals for the most part with obstetrical difficulties in the various forms of uterine duplication. Very little has been reported on the gynecological conditions arising therefrom. At the clinic of the Free Hospital for Women, Brookline, Massachusetts, 11 cases of bicornuate uterus have been encountered. Five of the 11 patients were single and nulliparous; 3 were married and nulliparous; 3 were married and had 4, 3, and 2 children respectively, each having had in addition two miscarriages. Seven of the series complained of dysmenorrhœa in none of whom was hæmatocolpos or hæmatometra found. Five of the whole group complained of menorrhagia and one of metrorrhagia. One patient menstruated regularly every 3 weeks and one every 2 weeks. Whether or not menstruation occurred alternately from either side of the double uterus is unknown.

At operation pelvic inflammation was found in 5 cases. The presence of a vesicorectal fold was recorded in only two. In 3 cases the pre-operative diagnosis had been of fibroid tumor. At operation a small fibroid only was found in one of these. Undiagnosed fibroids were found in 3 other cases. The ovaries were undescended in 2 patients. One patient with hæmatocolpos and hæmatometra had not complained of dysmenorrhœa. Three unilateral and three bilateral supravaginal hysterectomies were performed. One of the patients who had had a unilateral hysterectomy had a normal labor at a later date. Two patients had no abdominal operation; one was explored and two had uterine suspensions.

Seven cases were classed under 2a in Reed's table (uterus duplex sine didelphys, uterus bicornis); 2 were classed under 4 in the table, and 2 were didelphys uteri since there was complete duplication of body, cervix, and vagina.

Menstruation in a bicornuate uterus is as complete as in a single uterus, but it is heir to more pathological possibilities. All writers agree that dysmenorrhœa may be caused by retention in a rudimentary horn. In this series, however, no dysmenorrhœa could be attributed to retention. The diagnosis is based on the finding of an associated periodic swelling due to retention in the rudimentary horn when the menstrual flow is apparently normal in amount (unilateral hæmatocolpos due to atresia vaginalis lateralis). Dudley reported a case of intractable dysmenorrhœa with failing health in which he found a bicornuate uterus. Removal of both horns resulted

in a cure. McArthur described an operation for bicornuate uterus performed on a patient suffering from severe dysmenorrhœa. He cut the vaginal septum from below and then removed the remainder of the septum by the abdominal route joining the cut edges of the uterus as one would a rupture. There was complete recovery. In neither of these cases was hæmatocolpos or hæmatometra present.

Bainbridge stated that menstruation may take place every 2 weeks, first from one side, then from the other, the patient losing in all about as much blood as during a normal period. He stated also that one horn may menstruate while a pregnancy exists in the other. Among the 8 cases of uterus duplex reported in the records of the Boston Lying-in Hospital, no bleeding occurred during pregnancy. Moench said that while pregnancy exists in one horn, the other ceases to menstruate. Cleveland described the discharge of an anomalous decidual membrane during pregnancy in a case of bicornuate uterus. The membrane probably came from the unimpregnated horn.

Pregnancy in uterus duplex has been widely discussed. There was no indication that sterility occurred more often than it does in single uteri. A small majority in the literature and 6 of the 8 cases in the records of the Boston Lying-in Hospital were primiparæ. Numerous repeated pregnancies, even after unilateral hysterectomy, have been reported.

In cases of uterus duplex, abortions are common, the number depending probably on the frequency with which implantation takes place in the rudimentary horn. This is essentially an extra-uterine pregnancy. The ill developed horn cannot accommodate its rapidly growing physiological tumor, and abortion results. Rupture of one horn in the early months of pregnancy occasionally occurs, but abortion with expulsion of the products by the natural route is usual. Picot (2) reported a case in which there were 14 abortions, and Gautermann (2) cited a case in which three children were born from the right horn and nine abortions occurred from the left.

Hirst and Herman stated that it is impossible to make a diagnosis between tubal pregnancy and pregnancy in a rudimentary horn. Kelly and Noble, however, stated that the diagnosis may be made by the position of the round ligament, which in the latter is connected with the distal side of the tumor instead of with the proximal portion as in the former.

Pregnancy in bicornuate uterus is rarely interrupted between the third and the eighth month; frequently it goes on beyond term. If rupture occurs it is during the last month or at labor. Uneven distribution of uterine muscle and misdirected forces serve to protract labor and weaken the uterine wall, increasing the danger of rupture. In one of the Boston Lying-in Hospital cases, the head of the child protruded through a ruptured fundus.

There seems to be some difference of opinion among obstetricians regarding the extent to which a double uterus may complicate labor. Transverse positions are common and in primiparæ should lead one to think of this anomaly. Labor is long; pains are weak; intra-uterine death is frequent. Irregularity of the fetal heart is common, not only during labor but in the later months of pregnancy. Deformities of the fetus, such as scoliosis, talipes equinus and, oddly enough, polydactylism, are not infrequent. DeLee reported a difficult delivery in which the child straddled the septum of a partially septate uterus. Placentas are more frequently adherent and are often bipartite. A succenturiate lobe may be present. Findlay said that placental tissue is likely to be retained in the uterus and lead to infection and hæmorrhage.

In Falls' series of 15 cases 6 primiparæ were in labor over 17½ hours. Cæsarean section was performed on one of these after 41 hours of labor. Of the 8 Boston Lying-in Hospital cases, one was in labor slightly over 17 hours and was delivered by low forceps; one had a 10 hour labor and was delivered by mid-forceps; one went 48 hours and was then delivered by cæsarean section. In this case the uterus had just begun to split. Four of the remaining cases had cæsarean section. One of these had a ruptured uterus at 5½ months, having previously had four normal deliveries. The eighth case had a premature separation of the placenta and delivered herself of a 7 months' macerated fetus.

Falls estimated that the average blood loss is 500 cubic centimeters which is twice the normal average. Involution is usually delayed.

Operative measures are recommended by Rockey, McArthur, Strassmann, and Dannreuther, if the condition is diagnosed in nonpregnant women. Rockey clamps the upper and lower borders of the vaginal and uterine septum and leaves the clamps in place for 36 hours. He advocates the same procedure for bicornuate uteri, but he puts the patient in the Trendelenburg position and closes the clamp slowly to obviate the possibility of catching gut between the two horns. His patient has since had two normal pregnancies.

McArthur's operation is described above. Strassmann (15) in discussing Falls' paper proposed an operation for uniting the two horns, i.e., to incise the fundus from one to the other and then to join the two halves.

Dannreuther (15) in discussing the same paper stated that the bladder is frequently displaced upward because of its attachment to the rectovesical fold. He advised separating the fold well back in the sulcus, because if the patient ever comes to hysterectomy the transverse incision across the uterovesical peritoneal fold customary in hysterectomy would jeopardize the bladder wall.

Numerous odd cases are reported in the literature. In 1896 Cullen and Wilkins (25) reported a case of pregnancy in the rudimentary horn and collected 39 other cases. In 1900, Kehrer collected 84 cases. In 78 of these the proximal end of the rudimentary horn did not communicate with the uterine cavity, and pregnancy must have followed external migration of the spermatozoon or ovum. Caswell and Horn both reported cases of torsion of the pregnant half of a uterus didelphys. Caswell's case went through a normal pregnancy after partial hysterectomy.

Falk artificially aborted a woman with grave heart disease who was 2 months pregnant. She returned 3 months later with her uterus corresponding to a 5 months' pregnancy. This was due to another ovum situated in the other half of a bicornuate uterus.

Monat reported a case of a rudimentary horn in an inguinal hernia. He stated that there were only 37 on record and that a great majority occurred on the left side. He advised operating only to reduce the hernia.

A bicornuate uterus or uterus didelphys is the popular explanation for superfetation and superfecundation. Twin pregnancies are frequently reported in double uteri, and Ross reported a triple pregnancy. According to Bainbridge, Dibierre quoted an instance of a woman who bore one child on July 16, 1870, and another on October 31, of the same year, both full term. She had had three menstrual periods between the confinements. Jellinghaus reported one case of a full term child in one horn and a 4 months' fresh fetus in the other, and another in which the patient was delivered of a full term white child from the left horn and 2 months later of a full term black infant from the right horn. The father's race and ancestry were not included in the report.

SUMMARY AND CONCLUSIONS

1. The etiology of bicornuate uterus probably lies in some mechanical obstruction preventing complete union of the paired ducts of Mueller.

2. A bicornuate uterus is heir to the same gynecological conditions as a single uterus more frequently than to its unique tendency to dysfunction.

3. The obstetrician must be aware to the possibility of a dual organ and its potential catastrophes if labor is uncommonly slow, if the fetal heart is irregular and rapid before labor begins, in all malpositions, particularly in primiparæ and when there is a history of repeated abortions before the fourth month.

4. Operative procedures are advisable in view of possible future pregnancies.

5. Superfetation may attribute its success to bicornuate, septate, or didelphys uterus.

For permission to report the Free Hospital cases and for assistance the author wishes to express his thanks to Dr. William P. Graves at whose instigation this study was undertaken. He is grateful to the staff of the Boston Lying-in Hospital for permission to report their cases.

BIBLIOGRAPHY

1. Ancel and Villemin. Bibliographie Anatomique, xvi, 489.

2. BAINBRIDGE, W. S. Duplex uterus with multiple pregnancy. Am. J. Obst. & Gynec., 1924, vii, 285–288.
3. BARKLEY, A. H. Bicornuate uterus with report of a case. Kentucky M. J., 1927, xxv, 358.
4. BRUGH, B. F. Case of bicornuate uterus unicollis with rupture of granid horn followed by operation and recovery. West Virginia M. J., 1926, xxii, 636–637.
5. BURRAGE, W. S. Text Book of Gynaecological Diagnosis, 1910.
6. BYFORD. Manual of Gynæcology, 1895.
7. CASWELL, C. C. Am. J. Obst. & Gynec., 1926, xi, 853.
8. CATLIN, J. J. Double uterus. J. Am. M. Ass., 1924, lxxxii, 2030.
9. DANNREUTHER, W. T. Uterus duplex unicollis with comments on malformations of the uterus. Am. J. Obst. & Gynec., 1927, xiv, 376.
10. DAVIES, J. C., and CŒLLAN, C. J. Pregnancy alternating in horns of uterus didelphys. Brit. M. J., 1927, ii, 59–60.
11. DUDLEY. Principles and Practice of Gynæcology, 1913.
12. DURAISAMY, A. E. Twin pregnancy in bicornuate uterus. Indian M. Gaz., 1926, lxi, 497–498.
13. EDEN and LOCKYER. New System of Gynæcology, Vol. I.
14. FALK. Abstract of tolerance of pregnant uterus in artificial abortion in doubly pregnant uterus. J. Am. M. Ass.
15. FALLS, F. H. Study of pregnancy and parturition in primiparæ with bicornuate uteri. Am. J. Obst. & Gynec., 1928, xv, 399.
16. FINDLAY, P. Diseases of Women, 1913.
17. GUILLEMINET and MICHON. Abstract of ten cases of double uterus; clinical aspect and treatment. J. Am. M. Ass., 1924, lxxxiii, 1719.
18. GUY, W. P. Case of pregnancy in double uterus terminated by hysterectomy. Am. J. Obst. & Gynec., 1924, viii, 500–501.
19. GREENFIELD, B. H. Case of bicornuate uterus unicollis. Surg. Clin. N. Am., December, 1926, vi, 1695–1696.
20. HERMAN, G. E. Diseases of Women, 1913.
21. HINCKLE, W. A. Case of double anus, vagina, and uterus. J. Am. M. Ass., 1928, xc, 455–456.
22. HIRST, J. C. Manual of Gynecology, 1918.
23. HORN, H. W. Arch. Surg., 1924, ix, 606–612.
24. JACOBS, A. Am. J. Surg., 1928, iv, 500–507.
25. KELLY and NOBLE. Gynecology and Abdominal Surgery, 1910.
26. KERWIN. Uterus septus with special reference to operative technique. Am. J. Obst. & Gynec., 1925, ix, 369–375.
27. KICKHAM, C. J. Uterus septus duplex, with full-term twin pregnancy. Surg., Gynec. & Obst., 1922, xxxv, 443–444.
28. LAMB, J. Twin pregnancy in bicornuate uterus with contracted pelvis, delivery by cæsarean section. Brit. M. J., 1923, ii, 1152.
29. MCARTHUR, A. N. Med. J. Australia, December 21, 1918.
30. MILLER, N. F. Clinical aspects of uterus didelphys. Am. J. Obst. & Gynec., 1922, iv, 398–408.
31. MOENCH, G. L. Am. J. Obst. & Gynec., 1927, xiii, 60–64.
32. MONAT, T. B. Inguinal hernia containing rudimentary horn of bicornuate uterus. Brit. M. J., 1926, i, 237–238.
33. MUNDELL, J. J. Case of pregnancy in double uterus (uterus didelphys). Am. J. Obst. & Gynec., 1923, vi, 601–606.
34. NASH, W. G. Placenta prævia in double uterus, cæsarean section, recovery. J. Obst. & Gynæc., 1922, xxix, 644.
35. NEWTON, F. C. Uterus didelphys; notes on its developmental etiology and clinical significance. Ann. Surg., 1924, lxxix, 102–113.
36. PIQUAND. Rev. de gynéc. et de chir. abd., 1910, xv, 401–466.
37. POLAK, J. O. Life history of double uterus. New York State J. M., 1923, xxiii, 107–109.
38. PURSLOW. Case of hæmatocolpos, hæmatometra and hæmatosalpinx. J. Obst. & Gynæc., 1923, xxix, 643.
39. REED. Diseases of Women, 1913.
40. ROCKEY, A. E. Ann. Surg., 1916, May.
41. ROWLET. Report of a case of double uterus with pregnancy in each horn. J. Florida M. Ass., 1925–26, xii, 5.
42. TALLANT, A. W. Repeated cæsarean section in septate uterus. Atlantic M. J., 1926, xxix, 779–780.
43. WARDLOW, Y., and SMITH, W. P. Uterus duplex bicornis. Surg., Gynec. & Obst., 1922, xxxv, 497–500.
44. WILLIAMS, L. H. Case of uterus duplex with adenomyoma. J. Obst. & Gynæc., 1924, xxxi, 286–288.

THE RELATION OF MATERNAL PELVIC DISEASE TO DEFORMITIES IN THE NEWBORN

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ONE of the theories regarding the causation of monstrosities and other gross morphologic defects attributes them to disease of the generative organs. Mall believed that environmental conditions, interfering with the proper implantation of the ovum, were the chief contributing influences, and that endometrial disease was among the most important of these. Our intention is not to discuss here the relative validity of the different theories, but merely to present some recently collected observations on the relationship between pelvic disease and the formation of severe anatomical defects in the newborn child.

Pelvic disease with uterine hæmorrhage frequently renders a patient incapable of bearing children. It is, for this reason, difficult to secure records of any larger number of pregnancies in women with pelvic disease.

In a recent investigation (6, 7, 8) of the effect of pelvic radiotherapy on the health of subsequent children, we studied the records of a great many women who received roentgen or radium treatments for pelvic disease while non-pregnant, and who at a later date bore children. Most of these children were healthy while a small number were unhealthy or otherwise defective. The factors responsible for the ill health were readily determined in many cases. Irradiation was not believed to be the cause of the ill health or deformities in any of them. The question which now had to be considered was: Could the ovarian or uterine disease have caused the anatomical defects of obscure origin, exhibited by several of the children?

The large number of births, in women who had received therapeutic irradiation for pelvic disease, offered an excellent opportunity to study the relationship between such disease in the mother and malformations in the children.

A review has been made of the health reports of 310 children born of women suffering from pelvic disease. Approximately one-third of the mothers of these children suffered from idiopathic uterine hæmorrhage, and one-third had myomata. The remainder were treated for various other gynecologic conditions, such as adnexitis, amenorrhœa, and carcinoma of the cervix or vulva.

Among the 310 children mentioned above, 3 (1 in 103) were grossly deformed (Table I). Most of the remaining 307 were healthy, although several were unhealthy. The incidence of ill health was not believed to be greater than the usual incidence. The pelvic disturbances manifested by the mothers of the 3 malformed children are described in Table II.

In order to determine how the ratio of 1:103 compared with the birth rate of monsters in the general population, the statistics in Table III were assembled. Of the 95,366 children, 419 (0.4 per cent) were grossly deformed. The ratios for the various groups forming this total ranged from 1:111 to 1:456. A comparison indicates that pelvic irradiation has relatively slight influence upon the birth rate of monsters. If the diseases associated with uterine hæmorrhage had played an important rôle in the production of deformities, it is believed that the percentage of malformed children (0.97 per cent) born of the women with pelvic disease would have been considerably greater than that (0.4 per cent) in the general population.

In considering the relationship between pelvic disease and the birth of monsters, it must be borne in mind that many women with disease of the pelvic organs undoubtedly improve under irradiation treatment. The influence of such treatment upon the incidence of defective production, however, cannot be stated. Disregarding this complicating factor, it is believed that uterine disease is negligible as a factor in producing deformities in the offspring. The observations recorded here, although representing only a small group of individuals, strongly suggest that uterine

TABLE I.—INCIDENCE OF GROSS DEFORMITIES AMONG CHILDREN BORN OF WOMEN SUFFERING FROM PELVIC DISEASE AND TREATED BY IRRADIATION

Births	310
Malformed children (0.97 per cent)	3
Ratio	1:103

TABLE II.—TYPE OF PELVIC DISTURBANCE SUFFERED BY THE MOTHERS OF THREE DEFORMED CHILDREN

Author	Pelvic condition	Malformation
1. Coffey	Metrorrhagia	Anencephaly
2. Gummert	Myoma uteri	Microcephaly
3. Moeller	Menorrhagia	Hydrocephaly

TABLE III.—BIRTH RATE OF MALFORMED CHILDREN IN THE GENERAL POPULATION

Source of data	Births	Malformations	Ratio
Chaussier*	22,293	132	1 : 169
Puech*	778	7	1 : 111
Schworer*	39,917	88	1 : 456
Winckel	12,378	87	1 : 142
Winckel	20,000	105	1 : 190
Total	95,366	†419	

*Quoted from Marchand.
†0.4 per cent.

disease characterized by hæmorrhage does not increase the tendency to the production of malformations in the subsequent children. However, it is realized that endometrial disease may be an important factor in the production of abortion.

CONCLUSION

It is concluded from this study that uterine or ovarian disease apparently has no relation to the production of deformities in the newborn child.

BIBLIOGRAPHY

1. COFFEY, T. Personal communication to the authors.
2. GUMMERT. In discussion of Borell, Zur Frage der temporaeren Roentgen-sterilisierung und ihre Indikationsumgraenzung. Arch. f. Gynaek., 1925, cxxv, 607–608.
3. MALL, F. P. A study of the causes underlying the origin of human monsters. J. Morphol., 1908, xix, 3–367.
4. MARCHAND. Missbildungen. Eulenburg's Real-Encyclopedie der gesammten Heilkunde, 1897, xv, 439.
5. MOELLER, W. The effect and risks of the radium treatment in benign gynecological complaints. Acta obst. et gynec. Scand., 1925, iv, 222–242.
6. MURPHY, D. P. Ovarian irradiation, its effect on the health of subsequent children; review of literature experimental and clinical, with a report of 320 human pregnancies. Surg., Gynec. & Obst., 1928, xlvii, 201–215.
7. Idem. Ovarian irradiation and the health of the subsequent child; a review of more than 200 previously unreported pregnancies in women subjected to pelvic irradiation. Surg., Gynec. & Obst., 1929, xlviii, 766–799.
8. MURPHY, D. P., and GOLDSTEIN, L. The etiology of the ill health in children born after preconception pelvic irradiation. Am. J. Roentgen., 1929, xxii, 207-219.
9. WINCKEL, F. Ueber menschliche Missbildungen. Samml. klin. Vortr., Gynaek., 1903–1907, cxxxvii, 93–124.

CLINICAL SURGERY

FROM THE MAYO CLINIC

THE MANAGEMENT OF LESIONS OF THE POSTERIOR WALL OF THE DUODENUM[1]

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ULCERS of the posterior wall of the duodenum present a difficult surgical problem. Such ulcers are much more common than is generally recognized. The majority of lesions which are found in the duodenum at necropsy are on the posterior wall, and it is a common experience, in those operations which permit satisfactory exposure, to encounter one or more ulcers in various stages of development on the posterior aspect of the duodenum. The significance of such lesions, I believe, has not been sufficiently appreciated.

It is not an infrequent experience, in operating on a patient who has given a typical history of duodenal ulcer, to find that the anterior wall presents little evidence of an ulcer, and that on palpation, there appears to be no lesion posteriorly. However, in many such cases, if the duodenum is opened, and the posterior wall carefully examined under direct vision, a perforating ulcer is found on the posterior wall. The lesion may not have given signs of its presence to the roentgenologist and the diagnosis of peptic ulcer may have been made on symptoms alone. I have met with a number of instances in which there has not been any appreciable evidence of inflammatory reaction in the anterior wall and in which there has been a crater in the posterior wall which could not be identified in any other way than by visualization. In some of these, hæmorrhage had been the predominant symptom. Under such circumstances, the importance of visualizing the lesion cannot be overestimated, inasmuch as the prospects of immediate relief and protection against subsequent hæmorrhage depend, to a considerable extent, on the eradication of the lesion. Nevertheless, when a local procedure does not seem advisable because of technical difficulties, the curative value of gastro-enterostomy for hæmorrhagic duodenal ulcer, wherever it may be situated, should not be overlooked.

In other cases, the presence of an ulcer situated posteriorly is suggested by an obvious inflammatory process of the anterior wall, which is characterized by stippling on palpation, and thickening of the duodenal wall, without any characteristic scarring. In many cases, excision of the anterior inflammatory area will disclose a well defined crater on the posterior wall. Removal of an anterior ulcer, without visualization of the posterior wall, leads to error. In other types of cases, a well defined ulcer of the anterior wall with a distinct crater of considerable size may be associated with one or more other lesions on the posterior wall. It is hardly necessary to point out the importance of knowing whether or not a lesion exists in the posterior wall, since failure to identify such a lesion may easily explain some of the disappointing results which follow operations, particularly when the patient's history has been characterized by repeated hæmorrhage. A safe rule to follow, therefore, is that a lesion in the posterior wall should always be suspected when there is a history of bleeding.

When lesions are encountered in the posterior wall the surgical management will vary considerably. It has been my practice, when it seems desirable or feasible to carry out excision of a lesion in the anterior wall, first to open the stomach about 2 or 3 centimeters proximal to the pylorus, midway between the greater and lesser curvatures. The opening should be large enough to admit the finger and to permit careful exploration of the posterior wall of the duodenum. If a crater is found posteriorly, and if, from its size and distance from the pylorus, it seems unwise to attempt its removal, the opening made for the exploration is closed and the management of the lesion carried out by performing either posterior or anterior gastro-enterostomy. If the posterior crater appears to be excisable, the exploratory incision may be extended around the anterior lesion, with removal of sufficient tissue

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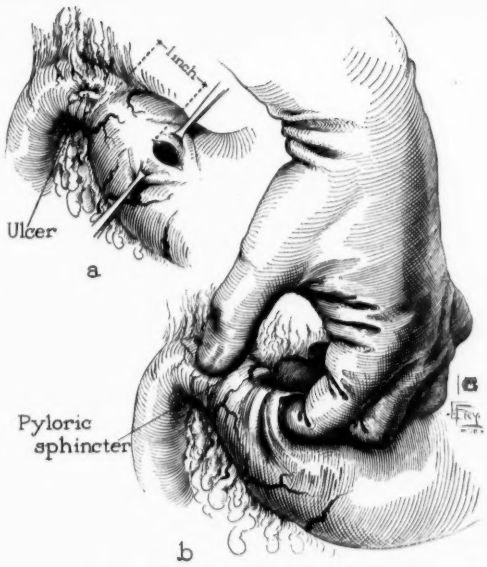

Fig. 1. Exploration of duodenum.

from the anterior aspect of the antrum, pylorus, and duodenum (the importance of which has been emphasized by Judd), so that in the subsequent reconstruction of the pyloric outlet a satisfactory lumen can be made.

The methods by which ulcer of the posterior wall of the duodenum may be dealt with are numerous. They are: (1) excision by cautery combined with pyloroplasty or gastro-enterostomy; (2) excision by knife, with reconstruction of the pylorus by the method of Heineke-Mikulicz, Finney, C. H. Mayo, Judd, or Horsley; (3) partial duodenectomy, by the Billroth I method or one of its modifications; and (4) partial duodenectomy and partial gastrectomy by the Billroth II method, the Pólya method, and so forth.

I wish particularly to draw attention again to a method of excision by cautery of lesions of the posterior wall of the duodenum. It is reasonable to assume that when a bleeding type of ulcer is present, it is desirable that it be removed. Unfortunately, the ulcer may be so situated, the duodenum may be so deeply placed, or a periduodenal inflammatory process may be so extensive, that excision is prohibited because of the risk entailed. In the majority of cases, however, satisfactory removal may be effected when the anterior aspect of the pyloric end of the stomach, the pylorus, and the duodenum have been widely opened and the posterior wall clearly exposed.

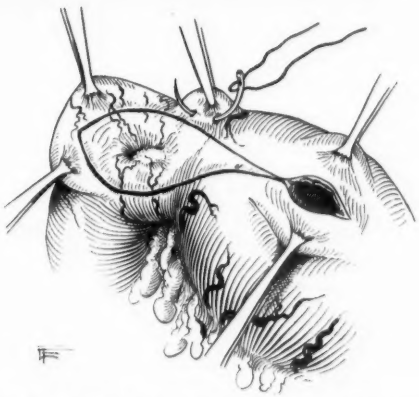

Fig. 2. Excision of ulcer on anterior wall of duodenum.

Then the ulcer can be dealt with according to its situation. If the posterior lesion is near the inferior or superior border of the duodenum, the incision can be continued around the lumen to include the ulcer and the resulting defect closed. This procedure is followed by reconstruction of the anterior defect. If, however, the ulcer is centrally situated and is distant from the pylorus, the cautery offers a safe and direct method of excision. If the full effect of the cautery is employed, the posterior lesion can be excised completely and the resulting defect can be repaired by sutures introduced from the mucosal side. In the majority of cases, following this procedure, the anterior defect can be reconstructed so that a satisfactory outlet of the stomach is obtained.

When the nature of an anterior lesion indicates the advisability of excision of a portion of the anterior wall, or when it is necessary to visualize the posterior wall because of the absence of any clear evidence of a lesion anteriorly, and when

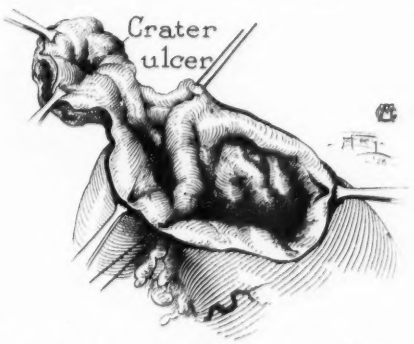

Fig. 3. Excision of ulcer on anterior wall of duodenum.

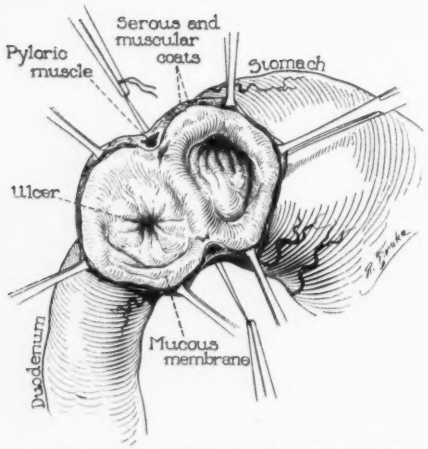

Fig. 4. Typical ulcer of the posterior wall of duodenum.

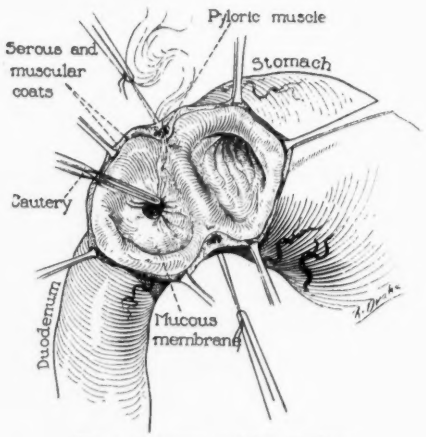

Fig. 5. Cautery excision of ulcer.

the history and roentgenographic evidence is positive for ulcer, the duodenum and pylorus are first mobilized to a reasonable extent, packs are carefully placed around the duodenum, and the liver is retracted so that adequate exposure of the entire first part of the duodenum is possible. The site of any inflammatory process in the anterior wall of the duodenum is marked off by Allis forceps, placed on the inferior and superior borders of the duodenum, slightly below the level of the inflammatory area. Similar forceps are placed on the greater and lesser curvatures of the stomach, about 3 or 4 centimeters proximal to the pylorus. The pyloric muscle is then deeply transfixed by two sutures, one near the greater curvature and one near the lesser curvature. The stomach is opened about 2 or 3 centimeters above the pylorus and any gastric content is aspirated by the suction pump. The finger is introduced through this opening, and through the pylorus, and careful exploration is made of the interior of the duodenum (Fig. 1). If it seems advisable to continue with the excision, the incision is carried down through the pyloric muscle to the inner side of the sutures already placed, the pyloric flap thus made is reflected, the mucosa of the anterior wall of the duodenum is inspected, and whatever lesion is present is excised by continuing the dissection in the uninvolved portion of the duodenal wall (Figs. 2 and 3). The two sutures of catgut which have been placed in the pyloric muscle can be used as tractors and the posterior wall can be satisfactorily exposed. It is most important to avoid any rough manipulation of the mucosa of the posterior wall, since it can easily be traumatized by the forceps, or even by a sponge, and the field obscured.

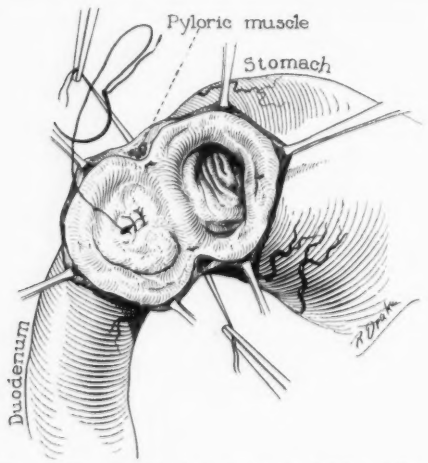

Fig. 6. Closure with chromic catgut of defect following cautery excision of ulcer.

It is in awkwardly situated posterior lesions that the particular value of the cautery is found (Fig. 4). When the base of the lesion has been cleansed by careful sponging, the point of the cautery is carried around the circumference of the lesion, until the mucosa is loosened and the cauterization of the base is continued until the ulcer is, to all intents and purposes, excised (Fig. 5). It is of considerable importance that such cauterization be done slowly, inasmuch as a low degree of heat is most effective. Such excision

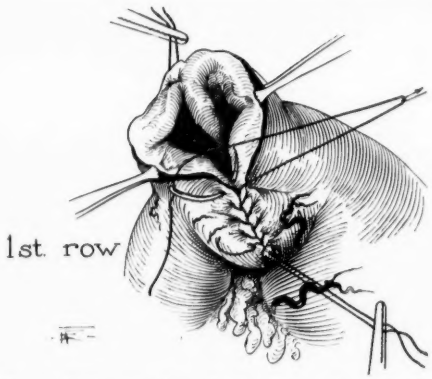

Fig. 7. Reconstruction after excision of ulcer of the duodenum.

is not associated with bleeding and the mucosal edges of the lesion are sufficiently excised by the cautery so that deep sutures of catgut can be used to obliterate the defect which has been made by the cautery (Fig. 6). Chromic catgut is the most desirable suture material to use, and the continuous figure-of-eight stitch is the most satisfactory method of closing the defect. When the lesion has been so dealt with, the opening in the anterior wall of the duodenum, pylorus and pyloric end of the stomach is closed in a transverse direction; the closure is made by continuous and interrupted sutures of catgut; and the upper and lower angles of the incision are reinforced by omental flaps (Fig. 7). The pyloric end of the stomach is sutured to the round ligament of the liver by sutures of catgut, as originally suggested by W. J. Mayo (Fig. 8). This procedure of dealing with an ulcer of the posterior wall has the

Fig. 8. Fixation of pyloric end of the stomach to the round ligament of the liver.

advantage of simplicity and efficiency, and in many cases seems the only reasonable direct procedure which can be used. In other words, it is often a question of either excision by cautery, or nothing. The results of operation have been very satisfactory for posteriorly situated ulcers associated with severe bleeding, and the mortality rate has been low. During the last 3 years in the clinic 52 lesions of the posterior wall have been dealt with in this manner, with one death.

FROM THE DEPARTMENT OF ORTHOPEDICS, NEW YORK POST-GRADUATE MEDICAL SCHOOL

TREATMENT OF UNUNITED FRACTURE OF THE NECK OF THE FEMUR[1]

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IN 1927, in order to compare the results achieved by the two operations which I have been employing for many years in the treatment of ununited fracture of the neck of the femur, I assembled a consecutive series of cases in which one or the other operation had been performed. Sufficient data were collected to enable me to report the results in 36 cases of the bone graft peg operation and 44 cases of my partial arthroplastic or reconstruction operation (1).

From this experience I learned again the value of reviewing one's results and correcting impressions by cold statistics. I learned a still more valuable lesson: that progression sometimes entails the necessity of changing one's mind. Previously I had favored the reconstruction operation and had committed myself to the statement that it was indicated in about 90 per cent of the cases seen late (2). I duly confessed my error and recanted. My subsequent experience, as I shall report it today, has only strengthened my determination to employ the autogenous bone peg operation in a larger percentage of cases.

THE BONE-GRAFT PEG OPERATION

I first described this operation in 1913 in *Murphy's Clinics*. The joint is exposed by an anterior incision straight downward from the anterior superior spine; a second is made over the great trochanter for the purpose of inserting the bone graft peg. The neck of the femur is inspected through the anterior incision. Eversion of the foot and limb causes the femoral fragments to separate anteriorly and the ends of both are then thoroughly freshened with osteotome and mallet. The foot is then restored to the anteroposterior axis and sufficient abduction (about 30 degrees) and traction applied, by means of the table, to bring the freshened fragment ends into close apposition.

Attention is next turned to the short incision over the trochanter which has been carried down to the fascia covering the vastus externus. These structures are now both split longitudinally so as to expose the lateral surface of the great trochanter. The point of application of the drill lies ½ inch below the bony ridge to which the fascia overlying the vastus externus is attached. Since the direction of the drill must follow the central line of the neck, due consideration must be given to the angulation of the neck to both the axis of the femur and the vertical intertrochanteric plane. In the average adult, the neck makes an angle of 130 degrees with the femur, and 12 degrees with the vertical intertrochanteric plane, when the foot is in the anteroposterior plane.

With the motor drill held in the direction thus indicated, a hole ½ inch in diameter is drilled through from the lateral aspect of the great trochanter to the broken end of the distal fragment. This point is determined by instrumental palpation of the head of the drill between the apposed fragments. The reading on the drill indicates the length of penetration through the distal fragment. With the drill head against the freshened end of the capital fragment, it is now carried into this fragment until the reading shows sufficient penetration. The degree of penetration is usually 7 or 8 centimeters (2½ inches) and is determined by a study of the roentgenogram. The drill is left *in situ* while a graft is taken from the crest of the tibia of the same side.

The tibia is exposed by a generous incision over its lower third. This lower portion is preferred on account of the greater thickness and strength of the cortex. A portion is chosen where the crest is straight and regular and the muscle and soft tissues dissected away. With my motor saw, a longitudinal cut is made on each side of the crest at a suitable angle with each other and an interval sufficient to provide a peg ½ inch in diameter after shaping (Fig. 3). Two transverse saw cuts are now made at an interval equal to the reading on the drill and the segment loosened by means of an osteotome and gentle blows of a mallet. The selected end of this segment is seized by two Ochsner clamps. The other end is inserted in the pencil sharpener cutter attached to the dowel-shaper, by means of which the end is shaped to a blunt conical point favorable not only for subsequent engagement in the dowel tool, but for reception in the drill hole already prepared in the femoral fragments. The pencil sharpener attachment is now replaced by the dowel tool and the

[1]Read before Pan Pacific Surgical Congress, Honolulu, August 23, 1929.

peg run through it. During both these shaping processes, a drip of normal saline is arranged to fall constantly on the tool not only to hasten its cutting but to relieve any possibility of undue heat. The saline solution also prevents dehydration of the graft by exposure to the air. Moreover, in the industries either oil or saline solutions are used in the cutting of hard substances, for the purpose of clearing debris from the path of the cutting instrument as well as for increasing the speed of cutting and for diminishing friction. The peg is now inserted into the drill hole in the trochanter (after removal of the drill) and driven home with the bone drift and mallet. With the end of the handle of a wooden mallet against the great trochanter, close to the peg graft and by means of blows of the palm of the hand or a sandbag against the head of the mallet, I attempt to secure close approximation of the fragments. The deep fascia is closed by interrupted sutures of No. 1 chromic catgut and the skin with a continuous suture of No. 0 plain catgut. The limb is put up in a posture of slight abduction, in a double plaster-of-Paris spica extending to the base of the toes on the affected side and to the knee on the sound side.

Let us examine in detail some of the points in this description of the operation. I have mentioned the necessity for consideration of the relation of the neck of the femur to the planes of the body. It was stated at a recent meeting of some of the world's most eminent orthopedic surgeons that the operation was "too difficult for any but the special few." I would be in agreement with that statement if it were impossible for all but the few to visualize the course of the femoral neck and to direct their operative procedure in accordance with the clear anatomical demands. The results which one eminent surgeon showed in roentgenograms of bone pegs that missed the capital fragment entirely or went clear through into the pelvis, would make one tremble for the future of surgery if it were to forsake so completely its foundation on the precision of anatomy.

Success in surgery depends more on anticipation of difficulties than on skill in overcoming the unexpected. By means of the graduated drill and palpation between the fragments, one can determine the point to which the drill hole ought to be carried. No phenomenal skill is required: all that is demanded is preparation, prevision, and precision.

Such unfavorable experiences have no particular point against the bone graft peg; they are equally applicable to pegs made of dead bone and to nails which I never advise, however.

Fig. 1. Unsuccessful use of nail in fracture of the neck of the femur. The destruction of bone has been so great that the nail has changed its course 40 degrees. Extensive cavitation of both fragments found at operation.

This surgeon's criticism of the bone graft peg, on the ground that it degenerated at the point of junction and finally disappeared, would have been disconcerting if I had not known his predilection for manual methods and if he had not made the casual admission that his opinion was based on experience with one solitary case.

It is my conviction, after mature study of both fresh and ununited fracture of the neck of the femur, that from the standpoint of mechanical immobilization the Whitman abduction method offers everything that can be desired. If it is properly applied it brings about ideal approximation of the fractured surfaces and provides ideal conditions for immobilization. If, after skillful and correct application of this method in fresh fractures, the fragments do not unite, the problem goes beyond mere mechanics and becomes a biological as well as a physiological one; the principal requirement being to bring blood supply to the point of non-union as well as a flow from one fragment to the other, or in other words a collateral circulation. Neither in fresh fractures nor in non-union is the use of nails justifiable. There is nothing they can accomplish in fresh fracture that the Whitman method will not. Since nailing is, at the best, scarcely more effective in immobilizing than Whitman's method, the latter, being non-operative, should have the choice. Even the method advocated by Smith-Petersen of using a nail with flanges cannot hope to supplant the Whitman method. In my opinion,

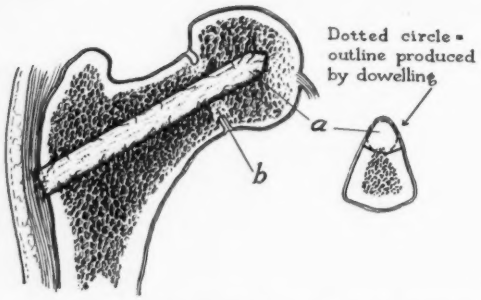

Fig. 2. Schematic sagittal section of bone graft peg in place in ununited fracture of the hip. Different shading of cancellous bone of head near ligamentum teres indicates bone of different texture because of preservation of vitality by blood supply through ligamentum tissue. The rest of the cancellous bone of the capital fragment shows evidence of ischæmia, which the bone graft peg when properly inserted overcomes by acting as a vascular conducting scaffold from the vascular cancellous bone of the distal fragment and the muscles overlying the distal end of the peg. *a*, Indicates haversian canals which are being canalized from these sources; *b*, indicates the callus which is built up between the femoral fragments and around the peg.

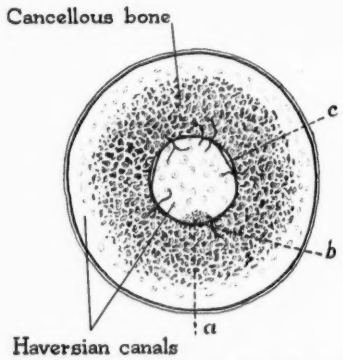

Fig. 3. Schematic cross section of bone graft peg in place in neck of femur. Shows same canalization (b) and consequent vascularization of the capital fragment and same blood supply to callus as is represented in sagittal section in Figure 2. *a*, Represents the cancellous bone at the endosteal angle of the crest of the tibia, *c*, represents osteogenetic portion of periosteum.

our waning faith in nails is not going to be restored by changes in the shape of the nail. The fault lies with the foreign body itself, as those of us who have been removing nails for years and observing their destructive effect on bone can emphatically attest.

The same argument holds in regard to ununited fracture. If Whitman's method, carefully and accurately followed, has failed, it is not sound surgery to expect some other, hardly more efficient, form of immobilization to be effective (Fig. 1). The history of the case demonstrates that something more than immobilization is necessary. Stimulation of osteogenesis is required, but still more vital is the nutrition and blood supply to the point of non-union and the anæmic capital fragment. This can be ensured only by the autogenous bone graft peg inserted by a most accurate fit. This can be accomplished only by the use of electrically driven automatic machinery which brings about a fit commensurate with that of a glass stopper in a bottle. The insertion of graft should not produce compression by too tight or inaccurate a fit nor should there be a dead space, filled with air, blood clot, or tissue debris, between the surface of the graft and the host bone tissues. In other words, there should be the closest coaptation of the haversian canals of the host and graft tissues so that early and complete canalization of the graft will take place.

A graft of irregular shaped cross section will not do. At the corners of the graft there will be compression resulting in both occlusion of host blood vessels and destruction of bone cells in both the graft and host tissues; at the flat or depressed surfaces of the graft there will be dead space filled with either air, blood clot, or tissue detritus produced by hammering in a misfit graft. In either instance the conditions are unfavorable as organization must take place before the blood vessels reach the graft, even if they do, thus resulting in either a delay or diminution of vascularization of the graft which may be fatal to the result. The old expression of a square peg in a round hole is a misfit here as in every other human endeavor and will never do. The early and complete vascularization which will occur only in an accurately fitted autogenous peg graft is not only essential to its survival but serves to carry blood and callus forming material to the anæmic capital fragment and to the point of fracture.

A further most important consideration is the fact that the closer the fit, the more accurate the work, the less callus to secure union is necessary.

In the hip there are peculiar conditions which militate against union even in a fresh fracture regardless of the constitutional condition or age of the patient:

1. There is little or no periosteum about the head and neck of the femur.

2. The synovial fluid, which tends to flow between the fragments as soon as fracture occurs, has an inhibiting effect on callus formation.

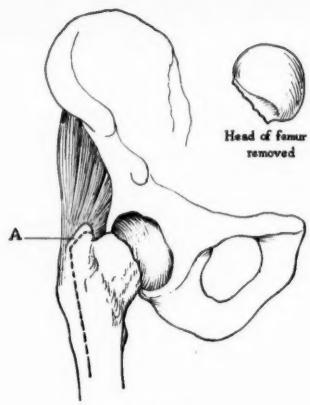

Fig. 4.

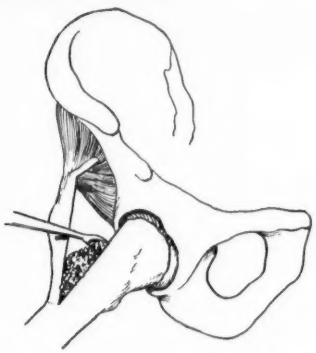

Fig. 5.

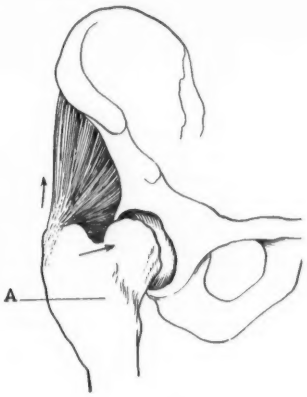

Fig. 6.

Fig. 4. Schematic drawing of author's reconstruction operation with removal of femoral head; dotted line indicates bone section of upper end of femur by broad osteotome.

Fig. 5. Displacement of upper end of bone muscle lever outward by abduction of hip which automatically thrusts the new femoral head into the acetabulum at the same time holding the bone muscle lever in oblique relation to the shaft. The angle is then filled with cancellous bone material from greater trochanter as indicated.

Fig. 6. Drawing from X-ray showing consolidation of union of bone-muscle lever with main portion of femur, angle being filled in *a*.

3. By all means the most important is the faulty blood supply. The neck and head have a meager blood supply on account of the absence of soft parts, through the attachments of which, in other areas than joints, circulation is supplied to the periosteum and bone. Even the fractured surfaces secure all their blood supply by way of minute blood vessels traversing the bone itself, there being no auxiliary supply from the surface.

It was shown by the extensive experimental work of Johnson that the rate of osteogenesis is in direct relation to the flow of blood to the zone of fracture. My clinical experience substantiates his conclusions.

If the fracture occurs in the central portion, the sole supply of the capital fragment is through the ligamentum teres. Even if the vessels in this structure are not damaged, the blood thus supplied is not adequate for even the normal vitality of the head, much less for osteogenesis and union.

The meagerness of the blood supply to the head of the femur was impressed on me by the study of the large number of capital fragments removed during the reconstruction operation. Phemister's experience has been similar. We have both observed much degeneration except in the zone immediately contiguous to the ligamentum teres and its vessels (Fig. 2).

Sir Arthur Keith, whose profound studies of bone growth are known to all, recently showed me some specimens at the museum of the Royal College of Surgeons to substantiate his belief in the early and remarkable vascularization of bone grafts. After the insertion of a bone graft peg through the fragments, the haversian canals are enlarged and canalized by new vessels. The cancellous bone at the endosteal angle of the tibial peg, though small in amount, also acts as a conducting scaffold for blood vessels which penetrate it from the vascular tissues overlying the trochanter and the vascular cancellous bone of the trochanter where the peg traverses it, and conducts them through the point of non-union and into the anæmic capital fragment (Fig. 2).

Campbell states that non-union increases rapidly with old age. This is dependent, I believe, not so much on general failure of recuperative power or diminution of osteogenetic power as on senile restriction of circulation.

AUTHOR'S RECONSTRUCTION OPERATION

For the reconstruction operation, the approach is that devised by Smith-Petersen, and is very similar to the Sprengel approach. The skin incision consists of two parts: (1) from the anterior superior spine directly downward, and (2) from this point over the anterior superior spine directly backward along the crest of the ilium for a distance of 3 or 4 inches. With a scalpel, the fascia lata is severed parallel with the crest of the ilium and sufficiently below it to leave a fascial flap to receive sutures in the approximation of

Fig. 7. Ununited fracture of neck of femur a few weeks after operation.

the severed fascia lata at the close of the operation. Then, with the wide osteotome used for splitting the spinous processes in the Pott's disease operation, the muscles are stripped down from the side of the ilium by subperiosteal separation, and are separated from one another directly downward from the anterior superior spine. The capsule of the hip joint is completely exposed. The joint is entered by a T-incision with the stem running directly downward along the neck of the femur. The head of the T is made about three-fourths of an inch from the rim of the acetabulum, for the purpose of furnishing a cuff of capsule to act as a lining of the outer portion of the joint, and for the neck to rest against when the head has been reconstructed. Care should be taken to make the incision into the capsule sufficiently spacious so that the difficulty of getting the head out of the acetabulum will be minimized. The ligamentum teres is then severed by means of a half-inch osteotome thrust deep into the joint, and any adhesions of the capsule to the periphery of the head are carefully separated with a scalpel.

The limb is then strongly everted by adjusting the Albee fracture table, so as to make room for the delivery of the head. With two long half-inch chisels or osteotomes, the head is pried out of the acetabulum, with a motion much like that used in eating with chopsticks; one osteotome is thrust into the inner and one into the outer substance of the head, and the two are used as levers against the soft parts to pry the head out of place. As soon as the head is delivered, the patient's limb and foot should be inverted by adjustment of the table, so that the foot points directly upward. Then, with the scalpel, the soft parts are severed in a straight line down on the anterior surface of the great trochanter, to admit the wide (1½ inches) osteotome that is to split off the lever as indicated in Figure 4. With this and a one-half inch osteotome, a lever measuring about 4 inches from the tip of the trochanter, including about one-sixth of the diameter of the shaft of the femur, but only a shell of bone at the tip of the trochanter, is separated and pried outward, producing a greenstick fracture at the lower end. The half inch osteotome is used to remove the shell from the superior tip of the trochanter with the muscle insertions. The second part of the bone incision at right angles with the first is made with the 1½ inch osteotome from above downward, care being taken not to separate the muscles and soft part from the bone while doing it. The last step is to fracture the bone muscle lever outward at its extreme lower end by using the wide osteotome before its removal as a lever (Fig. 5). Fragments of cancellous bone, removed from the cut surface of the trochanter and shaft by means of a curette, are placed in the angle of the gap thus formed (Fig. 5). The stump of the neck is then rounded so as to cause minimal irritation of the acetabulum. The assistant is directed to adjust the table so that the limb is brought to the limit of physiological abduction at the hip, and at the same time the upper end of the femur is lifted forward and guided into the acetabulum. Thus the bone lever is automatically held by the posture in the position shown in Figure 5.

The bone lever is pulled anteriorly and held with medium Kangaroo sutures in surrounding attached soft tissues. The wound is now ready for closure. All dead spaces are overcome by means of continuous suture of No. 1 chromic catgut. The skin is closed by continuous suture of No. 0 plain catgut. The line of incision and the suture holes are puddled with tincture of iodine, 3.5 per cent. The leg is put up in a long spica, extending from the tips of the toes to well above the costal margin, with the abducted position undisturbed. The plaster is molded so as to hold the upper end of the femur anteriorly and is kept on for a period of 7 weeks. The leg is then allowed gradually to resume the normal position. Patient is persuaded to begin walking with crutches immediately, and daily massage and manipulation, at hip and knee, are at once instituted.

I first performed this operation in 1915 and published the technique in 1919, several years before Whitman described his operation of similar nature. I still feel that it offers the best method of dealing with ununited fracture of the neck whenever erosion has produced such extensive loss of tissue of the neck that the bone graft peg cannot be applied.

It offers the following advantages: (1) It is the simplest operation yet devised and can be done in the shortest time. I have repeatedly performed this operation in 15 minutes, from the first incision to the last stitch. (2) Since the bone muscle lever displaced outward is only a shell of ¼ inch thickness at the trochanter tip, less shortening of the limb results from this method than by any other; in fact, the limb is lengthened by the operation in all cases, the exact amount varying with the existing shortening. (3) The erection of the bone muscle lever provides optimal mechanical conditions for the function of abduction and for the prevention of dislocation when the limb comes to the midline. The reconstructed upper end of the femur at the angle fills in with new bone and the lever becomes again incorporated with the shaft (Fig. 6). A case illustrating the unusual efficiency of the bone muscle lever was referred to the author some years ago by Dr. Crile.

A middle aged woman had consulted Dr. Crile, because of frequent dislocation and an entire absence of the function of abduction at the hip, following an arthroplasty for an ankylosed hip in which too much bone had been removed from the femoral head and neck. With the hip reduced, a longitudinal incision was made over the trochanter and a bone-muscle lever (Fig. 5) was formed. The operating time was 10 minutes. The result was excellent inasmuch as the hip never dislocated again and the function of abduction was most satisfactorily restored.

Fig. 8. Same some months later showing complete union with bone graft thoroughly consolidated with the fragments.

RESULTS

My 1927 report included a certain number of recent cases. The condition in each was described by way of an interim report. A definite opinion can now be given regarding these cases. The results in the entire group (including those reported in 1927) are set forth in Tables I and II.

It will be observed that in both groups the greatest number in any decade was between 50 and 59 and that half the patients were over 50. The bone graft peg, as might be expected, was favored over reconstruction in younger patients, but my experience has not shown that union is any less likely by this method in old age.

The time since operation was from 1 to 18 years. In the entire series of 228 cases the result may now be considered definite, since I have taken the reasonably conservative attitude of including no cases observed for less than one year.

Following the bone peg operation, the result was excellent in 90 per cent of cases; and after the reconstruction operation excellent in 78 per cent. If to these are added those cases in which the result was fair, the percentage of favorable results becomes 97 for the bone graft operation and 90 for the reconstruction. There have been no fatal cases since 1927, so that the mortality stands at 2 cases, 2 per cent of the reconstruction cases, or ½ per cent of the entire series, no death having occurred in the bone peg cases.

Notes are appended to the tables indicating the causes of the unfavorable results. Failure of the patient to co-operate during the postoperative treatment and convalescence stands out as the important cause of ill success. The patient is a free agent at all times and occasionally cannot be completely controlled.

I wish to emphasize that, as the tables show, union will follow the application of the bone peg in a large percentage of cases, when workmanlike methods are used with due regard to the principles of physiology and biology as well as those of mechanics (Figs. 7 and 8). The percentage in which union occurred is still higher than the 90

TABLE I.—END-RESULTS IN ONE HUNDRED SIXTEEN CASES TREATED BY AUTOGENOUS BONE GRAFT PEG.

Age at operation, in years:	Cases
0–19 years	2
20–29 years	11
30–39 years	16
40–49 years	22
50–59 years	38
60–69 years	27
Years since operation:	
1– 1½	30
2– 4	41
5–10	36
10–12	9
Results:	
Excellent	104
Per cent	90
Fair	9*
Per cent	7
Poor	3†
Per cent	3
Death	6

*In three cases the result was impaired by arthritis; in one the peg broke and reunited and in one motion was unduly limited.

†In both the peg broke, in one case due to failure of co-operation and excessive weight of the patient. One patient refused further treatment; the second returned for reconstruction 2 years later with ultimate excellent result. (Table II).

TABLE II.—END-RESULTS IN ONE HUNDRED TWELVE CASES TREATED BY AUTHOR'S PARTIAL ARTHROPLASTY OR RECONSTRUCTION OPERATION.

Age at operation, in years:	Cases
0–19	1
20–29	4
30–39	9
40–49	27
50–59	37
60–70	34
Years since operation:	
1– 1½	31
2– 4	49
5–14	32
Results:	
Excellent	87*
Per cent	78
Fair	14
Per cent	12
Poor	9†
Per cent	8
Death	2‡
Per cent	2

*Including case referred to in footnote in Table I.

†One case was complicated by malignant disease; in two cases patient did not co-operate in postoperative treatment; in two cases, although union was present, pain persisted, associated in one with flattening of the head.

‡In both cases within one week of operation; in one from chronic nephritis.

per cent in which an excellent result was obtained. The graft always united and in only 2 cases did it break and fail to unite.

Too much emphasis cannot be placed upon the importance of postoperative surveillance and care, particularly as to frequent X-ray studies of the graft and the filling in of the fracture hiatus with well consolidated callus. Stereoscopic exposures taken in different planes are necessary. Great care must be taken not to allow weight bearing until firm union is shown.

I feel constrained to take issue with the statement that the autogenous bone graft operation requires two operative teams. My average time for taking a graft from the tibia and shaping it into a peg is 6 minutes. Such delay is not of serious moment, but the increased risk of infection and the confusion of surgeons and nurses about a patient, when two teams are working, do not make for good surgery. In my opinion, such methods are to be condemned on the old fashioned principle of the cooks and the broth. Presumably it is shock that is feared, but I have not found this a serious consideration, as the absence of mortality due to this operation shows.

It was my expectation, after my study in 1927, to employ the bone peg operation and the reconstruction operation about equally. My recent statistics show that since then I have performed the bone peg operation in more than 50 per cent of the cases.

The choice of operation is determined by the condition of the fragments, and this depends on the previous treatment of the case as well as on the co-operation of the patient in that treatment. In many border line cases the choice of operation cannot be determined until the head and neck of the femur have been exposed. In some instances, the capital fragment may appear in the X-ray picture to be sufficiently long to receive satisfactorily a peg graft, but upon exposure the fractured end will be found to have undergone erosion by excavation and the head to be a mere shell. The principal causes of this condition are undoubtedly inadequate blood supply plus unwarranted locomotion. In a few other cases I have found much connective tissue both about the periphery of and in the joint resulting in limitation or absence of motion of the head in the acetabulum. In the event of any of these conditions being found, the arthroplastic operation rather than the graft peg is chosen. If all cases of fresh fracture are treated with alertness and skill, the number requiring either operation will be

materially reduced. If union fails and operative measures are carried out as early as possible and before serious erosion has occurred, the bone peg operation will be found feasible in practically all cases.

There are still too many cases in which faulty treatment of the original fracture, hasty conclusion as to the establishment of union, too early permission of locomotion, and delay on the part of the patient produce conditions that compel recourse to arthroplasty or reconstruction.

SUMMARY

1. In the event of failure of union in a fresh fracture of the neck of the femur following the Whitman method, the problem no longer remains a purely mechanical one, and therefore the insertion of foreign bodies in the form of boiled bone, nails, or screws is not indicated.

2. Because of inadequate blood supply not only does callus fail to form but the neck of the femur melts away. The principal indication, therefore, is to bring blood not only to the anæmic femoral neck and head but to the point of fracture. This is best accomplished by the cabinet-maker fitted autogenous bone graft peg.

3. The active osteogenesis associated with fresh fractures has subsided. In this respect, therefore, the indication is to supply an element capable of osteogenesis on its own behalf and this the autogenous bone graft peg does.

4. The survival of the graft, both as a vessel conducting scaffold and as a callus forming element, depends largely upon complete immobilization of it in both fragments but also of each fragment with the other. Mechanically the machine fitted peg graft fulfills this requirement in the superlative degree, at the same time that metabolism being stimulated by withstanding stress because of Wolff's law.

REFERENCES

1. ALBEE, FRED, H. Late end results in ununited fracture of the neck of the femur treated by the bone peg or the reconstruction operation. J. Bone & Joint Surg., 1928, x, 124–141.

2. Idem. Reconstruction and plastic operations on the hip. J. Am. M. Ass., 1925, lxxxv, 1345–1350.

SUPERNUMERARY KIDNEY

Report of a Case with Review of the Literature[1]

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From The Presbyterian Hospital

THAT true supernumerary kidney is probably the rarest of the congenital renal anomalies seems to be the uniform opinion of clinicians as well as of the authors of various textbooks. Congenital anomalies are being recognized in a much greater number of instances today than formerly, thanks to the widespread, careful urological study with modern diagnostic methods, and the result is an extensive literature dealing with the various congenital anomalies of the kidney.

Although the literature on congenital anomalies is extensive, the actual number of cases of true supernumerary kidney reported is exceedingly small. As a matter of fact, a review of the literature has revealed only 29 cases and this number with the case presented in this paper brings the number available for study up to 30. No doubt, this may not represent all the cases that have been reported; a few may have been overlooked, for a great deal of confusion exists in the literature today as a result of carelessness in selecting titles for case reports. Thus, for example, we find reports of accessory kidneys and supernumerary kidneys which are really double kidneys; in a few instances, horse-shoe kidney has been reported as supernumerary kidney, and some of the older descriptions of four kidneys would indicate horse-shoe kidneys. Moreover, some cases of supernumerary kidney have been found in articles, the titles of which would not suggest this rare anomaly.

True supernumerary kidneys are by no means of recent discovery. Blasius, writing upon this subject in 1677, quotes Martius who in 1656 described a human subject having two kidneys on one side and a single kidney on the other. The kidneys were of the usual structure and form. However, the kidneys on the side having two were smaller than normal. Each had its separate blood vessels branching from the aorta and vena cava and each its separate ureter. Hyrtl reported a case of true third kidney in 1841. Although he says the cases of four and five kidneys mentioned by the ancient authors are not all authentic, nevertheless he cites references to cases in which four and five kidneys were found. A brief historical review is to be found in the articles by Rayer and also by Voigtel.

This condition is not limited to the human. Pieth, an inspector of butcher's meat, found a third kidney in the iliac region in a fat cow.

Several years ago I reported a case of true supernumerary kidney and I now wish to add a second case of this rare and interesting anomaly.

CASE REPORT

Mrs. H. P., aged 22 years, referred by Dr. R. Drostenfels, was admitted to the Presbyterian Hospital on July 16, 1928. About a week ago she began to have severe pain in the region of the right kidney. The pain was so excruciating that she could not keep from weeping; it bore no relation to eating, deep breathing, urination or bowel movements; it radiated down the right side to the bladder region. There was some tenderness just below the right costal margin. Hot applications gave relief. The patient believed that the present illness followed a cold in the head at which time she had headaches, chest pains, blood-tinged sputum, malaise, and general body aches. For the past 7 days patient had been extremely weak, tired, and fatigued. The bowels had been constipated; cathartics gave no results. Since the onset of the present illness she had been obliged to void every 10 or 15 minutes and there had been present a sharp, intermittent pain over the bladder, associated with a desire to urinate. Pain was worse after urination and remained localized in this region. Associated with frequency there was burning after urination and this was located in the urethra and vagina. Since the onset of the present trouble the patient had been obliged to void several times at night, and urination was associated with burning and pain over the bladder region. Chills and fever had been present for one week; the temperature reaching as high as 103 degrees.

General physical examination was negative.

Examination of the abdomen disclosed a large hard mass on the right side which extended to the left of the umbilicus, upward under the arch of the ribs, and downward about one fingerbreadth below the anterior superior spine. The tumor was smooth, quite tender, and did not move on inspiration.

Cystoscopic examination was made July 17, 1928. Clear, sparkling urine was obtained, leading one to suspect a block on the right side. Right and left ureteral orifices were normal and there was no accessory ureteral orifice seen. Both ureters were catheterized without difficulty or obstruction. Urine obtained at this examination showed the following:

	Leucocytes per cu. mm.	Smears	Cultures	Tubercle bacilli
Bladder	2	No organisms	Sterile	None
Right kidney	160	No organisms	Sterile	None
Left Kidney	80	No organisms	Sterile	None

[1] Read at the annual meeting of The American Association of Genito-Urinary Surgeons, Manchester, Vermont, June 13, 14, and 15, 1929.

Phenolsulphonephthalein test was made and the dye appeared in the urine in 5 minutes. The output was:

	Per cent
First 30 minutes	25
Second 30 minutes	20
Third 30 minutes	15
Fourth 30 minutes	10
Total 2 hours	70

Guinea pig test showed the bladder and the right and left kidneys negative for tuberculosis.

Roentgen ray examination was negative for stone. A large amount of gas was seen in the colon and there seemed to be a mesial displacement of the colon on the right side.

A pyelogram of the right kidney taken July 17, 1928, showed a rounded soft parts shadow filling the right side of the abdomen from ribs to iliac crest deviating the ascending colon to the left. The right catheter curved outward to the lateral margin of the abdomen and upward to the costal border. The right pyelogram lay close to the lateral margin of the abdomen at the level of the second lumbar. The pyelogram was rotated so that kidney pelvis pointed laterally and the calyces mesially, except the superior calyx which was long and narrow and curved upward and outward to the tenth intercostal space. Other calyces and kidney pelvis were normal (Fig. 1).

The left catheter was in normal position.

Blood Wassermann test was negative. Blood chemistry examination showed: Urea, 21.8; uric acid, 3.0; creatinin, 1.5; non-protein nitrogen, 45.5.

Urinalysis, July 18, 1928, showed albumin and 3,500 leucocytes per cubic millimeter.

Temperature July 18, 1928, was 104 degrees.

Operation was performed July 18, 1928, under ethylene anæsthesia. The usual oblique kidney incision was made through the skin, fascia, and muscles. A tumor mass was exposed, which fluctuated, and was about the size described in the physical examination. This mass was located behind the peritoneum and behind and below the right kidney, extending upward to the diaphragm and downward below the iliac crest, medially to the vertebral column.

The right kidney was in front of, and rested on, this tumor. The right kidney was freed from the swelling and surrounding tissue and *found to be distinctly separate from this mass*. A trocar was introduced into the tumor mass and purulent fluid obtained which had the odor of urine. About 3 pints of fluid were aspirated at which point the patient suddenly went into collapse. The sac was sewed into the incision, a drain inserted, and the usual closure was made.

The temperature became normal on the fourth day after operation but gradually rose again reaching 105 degrees, the patient being extremely ill. Many cystoscopic examinations were made, and each time two normal looking ureters in normal positions were seen. There was no sign of an accessory ureter.

Further roentgen-ray studies were made on July 26, 1928. The patient was cystoscoped, both ureters were catheterized, and double pyelograms were made. After the ureters were catheterized and while the right ureteral catheter was being injected with bromide solution, bromide solution was injected into the rubber drainage tube that led into the infected hydronephrotic sac.

Examination of the films showed the following: on the left side the pyelogram was normal and the ureter was in its normal course (Fig. 3). On the right side were seen two pelves. The normal one was obtained by injecting the bromide solution into the right ureteral catheter. The second pyelogram, obtained by injecting the bromide solution into the drainage tube, showed a large hydronephrotic sac and a large dilated ureter that could be followed as far as the brim of the pelvis (Fig. 4).

A second operation was done September 7, 1928, under ethylene anæsthesia. The previous incision was reopened and the muscles separated. Dissection was carried around the large hydronephrotic sac. The ureter came into view, was followed down to the brim of the pelvis, at which point it was as large as my thumb, then ligated, cut, and carbolized with carbolic acid. The hydronephrotic sac and ureter were removed and the cavity packed with iodoform gauze because of oozing.

September 22, 1928. Our records show that enormous amounts of thin, straw-colored fluid were seen in the wound and on the dressings. This discharge was probably urine. Patient was given some methylene blue capsules and blue fluid was discharged from the wound. It was therefore assumed that the patient had an injury to the ureter of the corresponding kidney on the right side. The discharge of urine continued on this side. The patient later developed severe pain in the right side, swelling, chills, and fever. A diagnosis of infected kidney on the right side was made and nephrectomy advised. This advice was not heeded by the patient until January 7, 1929, at which time a second right nephrectomy was done. The kidney was very much enlarged with many adhesions around it, and here and there abscesses were seen in the kidney substance. The pelvis was hydronephrotic, and the entire wound showed the presence of an enormous amount of scar tissue. Convalescence was uneventful.

TABLE OF AGES

	Cases
9 months	1
10 to 19 years	1
20 to 29 years	10
30 to 39 years	5
40 to 49 years	2
50 to 59 years	2
60 to 69 years	2
Not stated	7
Total	30

TYPE OF CASES REPORTED

An analysis of the 30 cases showed that 21 were clinical cases, 8 were discovered at autopsy, that is, during the performance of an autopsy this renal anomaly was found, and the type was not mentioned in one. Of the clinical cases 2 died after operation, one in 8 days and the other in 2 months. Autopsies were performed. Many of the early autopsy cases, unfortunately, are not reported in detail so that much valuable information is lacking; hence they are of doubtful value for statistical purposes.

AGE

In 7 cases the age was not mentioned. The youngest case reported was 9 months old (Pick, quoted by Neckarsulmer) and the oldest 67 years (Cobb and Giddings). A review of the table of ages reveals rather striking facts in that the

SUMMARY OF THIRTY CASES OF

Case No.	Author	Type of case	Age	Sex	Side	Location	Course of ureter	Cystoscopic examination
1	Blasius				R	Below other kidney	Independent	
2	Calabrese	Clinical	55	F	L	Directly below left kidney	Not determined	Showed only 2 ureteral openings into bladder
3	Camiña	Clinical		F	R	Near vertebral column, at level of crest of innominate bones	Not fused	Two orifices in bladder, a third in vulva in proximity of urinary meatus
4	Cheyne	Clinical	22	F	R	Behind the peritoneum resting on the right side of the lower lumbar vertebræ and brim of pelvis	Not traced	None
5	Clifford	Autopsy. Death from pulmonary tuberculosis		M	R	Two kidneys buried in fat in close apposition to vertebral column, opposite junction of second and third lumbar vertebræ	Ureter of smaller ends in blind sac beneath mucous membrane of bladder between ureteral and urethral openings	None
6	Cobb and Giddings	Clinical	67	M	L	Retroperitoneal. Beside the inferior vena cava	Ureter not found	None
7	Depage	Clinical	20	F	R	In front of vertebral column, between the aorta and vena cava	Course not determined	None given
8	Dixon	Autopsy	45	M	L	Below usual kidney, partly in left iliac fossa, partly on quadratus lumborum muscle	Passed upward to join ureter of upper kidney	None
9	Fischer and Rosenloecher	Clinical (autopsy)	64	M	R	Below usual kidney, which was "high"	Medial from regular ureter was an enlarged ureter; course spiral, entered bladder in a fold of mucous membrane immediately above right ureteral orifice. Opening very small	Right ureteral orifice slightly swollen; left normal
10	Gayet	Clinical	21	F	L	Below usual kidney	Not traced	Two openings on left ½ cm. apart; similar arrangement on right

SUPERNUMERARY KIDNEYS

Pathology in accessory kidney	Pathology in other kidney	Duration of symptoms	Symptoms	Palpable tumor	Pre-operative diagnosis	Operation	Result
Ptosis	Ptosis	2 months	Attacks of pain in epigastric region. Vomiting	Yes	Nephroptosis and probable abnormal kidney	Nephronexy in both left kidneys	Pains disappeared.
Not given	Not given	Not given	Not given	Yes	Supernumerary kidney	None	
Distinctly lobulated	None	"Sometime"	Abdominal pain, indigestion, and hysterical condition; with severe pain, diminution in quantity of urine	Yes	Movable kidney (?)	Exploratory only	Cure.
Rudimentary, on section no differentiation into cortical and medullary substance	Fetal kidney not functioning						
Papillary cystadenoma	None	2 years	Sudden, severe pain followed by weakness. Tumor painful when bladder was full	Yes	Tumor, probably pancreatic cyst	Complete removal of growth	Good recovery.
Malpighian glomeruli less than normal	Increased in size, situated lower than normal	4 years	Lancinating pains at level of stomach	Yes	Tuberculous ganglion of the mesentery	1. Accessory kidney disengaged to the pedicle; section. 2. Decortication. Suture	1. Recurrence of pain; increase in size of kidney; painfulness on pressure. 2. Painful symptoms diminished but did not completely disappear.
Not given	Normal	Not given	Not given	Not given	None	None	
Pyonephrosis	Normal	4 months	Burning sensation in urethra; strangury	Yes	Division of ureter with two renal pelves, one pyonephrotically diseased	Nephrectomy	Normal recovery until eighth day. Collapse, death.
Small but normal in structure	Large pyonephrotic kidney		Pains in left kidney with pyuria. Subtotal hysterectomy with removal of intraligamentary dermoid cyst. After 3 weeks uninterrupted recovery, sudden painful crisis with tension in the kidney	Dermoid cyst, not kidney	None	Nephrectomy	Recovery.

SUMMARY OF THIRTY CASES OF

Case No.	Author	Type of case	Age	Sex	Side	Location	Course of ureter	Cystoscopic examination
11	Holmes	Clinical	30	F	R	Almost transverse over brim of pelvis close to promontory	Ureter divided into several branches near kidney with separate implantations on surface	None
12	Hyrtl	Autopsy	Middle aged	F	L.	At entrance to true pelvis, to left, on sacro-iliac symphysis, behind broad ligament	Beside usual ureter to fundus of bladder. Not fused	"A line inward" from orifice of usual ureter
13	Isaja	Clinical	27	F	R	To right of vertebral column	Fused with usual ureter 5 cm. from supernumerary kidney	Two ureters in normal site
14	Israel	Clinical	32	F	L.	Partly above and behind left kidney, which was slightly lower than usual position	Not fused	Two ureters entered bladder at normal points. Third orifice not found, probably in cyst of vagina
15	Kretschmer	Clinical	27	F	L.	Below	Ureter fused with that of normal kidney outside bladder	Small polyp on internal urethral orifice Mucopus. Two ureteral orifices only
16	Kuksinskaja	Clinical	43	F	R	Adjacent to adipose capsule of usual kidney	Not traced	Two normal ureteral orifices
17	Lebedeff	Clinical	19	F	L.	At the level of the bifurcation of the abdominal aorta	Independent ureter	None given
18	Linberg	Clinica	26	M	L.	Immediately aboveleft common iliac artery on the psoas magnus	Fused. Five canaliculi from accessory kidney joined normal ureter	Two orifices, normal location
19	Mangeti	Autopsy		M	L.	Above the normal kidney	Ureter lacking	
20	Maximovitsch	Clinical	39	M	L.	In true pelvis with hilus turned upward	Fused with ureter of usual kidney just before reaching bladder	Two normal orifices
21	McArthur	Clinical	Middle aged	M	R	Above the iliac crest	Joined usual ureter 2 inches below usual kidney	None

SUPERNUMERARY KIDNEYS—Continued

Pathology in accessory kidney	Pathology in other kidney	Duration of symptoms	Symptoms	Palpable tumor	Pre-operative diagnosis	Operation	Result
Extremely disorganized by chronic inflammation and fibrosis and secondary pyogenic infection	None noted	20 months	Attacks of pain, nausea, vomiting	Yes	None	Nephrectomy	Eventual cure.
None	Not given	Not given	Not given	Not given	None	None	
None	Smaller and lower than normal	21 years	Painful attacks at 2-year intervals accompanied by bilious vomiting. General weakness for 6 months. Weight and pain in abdomen accentuated after eating and fatigue. Obstinate constipation	Yes	Mesenterial tumor	Ptosed colon replaced	Cure.
Pyonephrosis	Posterior surface in upper portion showed a shallow impression due to pressure of the accessory organ	2 years	1. Pain in bladder, suppurating discharge from vagina. Left kidney painful. Vagina cyst. Fever 2. Purulent discharge from lumbar fistula	Yes	1. Suppurative perinephritis 2. Third kidney	1. Organ, regarded as left kidney, opened and drained 2. Nephrectomy	1. Alternating purulent discharge from vagina and lumbar fistula. 2. Eventual recovery.
Calculus. Small	Calculus	2 years	Recurring attacks of kidney stone colic associated with nausea and vomiting. Stones passed. Pain and burning on urination. Tendency to constipation. Urine turbid during last month	None	Incrustations of renal pelvis	Usual and supernumerary kidney removed	Cured.
Small kidney, with a cyst upon the convex edge	None	4 months	Tumor rapidly increasing in size	Yes	Tumor or cyst of right kidney	Nephrectomy	Recovery.
Somewhat smaller than normal			Uncomfortable and sometimes painful feeling in left lower abdomen	Yes	Dermoid cyst of ovary and pregnancy	Nephrectomy	Smooth recovery.
None	None	3 years	Pains in left abdomen	Yes	Retroperitoneal tumor	1. Exploratory 2. Nephrectomy	Cure.
Small and undeveloped							
None	Calculus in pelvis of left kidney. Right lower than normal	8 years	Pains in left lumbar region; strangury. Frequent micturition; cloudy urine	None	Calculus of left kidney	Calculus removed by pyelotomy	Cure.
Rudimentary. Small calculus	Calculus		Right renal colic, hæmaturia, purulent urine. Gravel passed	None	Calculus	Calculus removed. Nephrotomy	Cure.

SUMMARY OF THIRTY CASES OF

Case No.	Author	Type of case	Age	Sex	Side	Location	Course of ureter	Cystoscopic examination
22	Munro and Goddard	Clinical	23	M	L	Entirely without the peritoneum, close beneath the median line, somewhat to left	Passed to bladder apparently at the fundus	"Careful search for proximal ureteral stump proved fruitless"
23	Newman	Autopsy (death from malignant disease of ascending colon)	Adult	M	L	Close to upper margin of usual kidney	Fused with left ureter half an inch below pelvis of left kidney	
24	Pick (Neckarsulmer)	Autopsy (death from measles and pneumonia)	9 mo.	F	L	On upper pole of left kidney	Parallel to other ureter ending blindly in bladder in median direction from but very near left ureteral orifice	None
25	Rutschinsky	Clinical (autopsy)	28	M	L	Below normally located kidney	Not traced	(Autopsy.) Two ureteral orifices
26	Samuels, Kern, and Sachs	Clinical	29	F	R	At upper pole of right kidney	Passed posterior to blood vessels and pelvis of usual kidney. Orifice in anterior wall of vagina	Urethral and ureteral orifices normal
27	Marine Hospital Reports. See Reference No. 27	Autopsy (death from exhaustion due to laryngeal phthisis)	39	M	L	In hilum of left kidney	Pervious for 2.5 cm. from pelvis	None
28	London Medical Gaz. See Reference No. 28	Autopsy (death from typhus fever)	30	M	R	On right iliac artery and the psoas muscle	Right ureter passed down as far as the bifurcation of the aorta, to the third kidney; it passed through a longitudinal furrow in the supernumerary kidney and joined the ureter of the latter	None
29	Von Hanseman	Autopsy (death from perforated ulcer of the stomach)	57	M	L	Below usual kidney	Fused with usual ureter	None
30	Kretschmer. (Case reported in this paper)	Clinical	22	F	R	Behind the other right kidney	Fused with the other ureter outside the bladder	Two normal ureteral orifices

SUPERNUMERARY KIDNEYS—Continued

Pathology in accessory kidney	Pathology in other kidney	Duration of symptoms	Symptoms	Palpable tumor	Pre-operative diagnosis	Operation	Result
Hydronephrotic kidney with atrophy of renal tissue and, in places, tubular hyperplasia	Normal	20 years 4 months	Indigestion, abdominal pain, vomiting, fever Headache, fever, malaise, constipation, dull bearing-down pains over bladder	1. Yes 2. Yes	1. None 2. Not given	1. Aspiration of cyst-like tumor. It was opened and packed with iodoform gauze 2. Cyst removed	1. Discharging sinus. 2. Recovery.
Healthy renal tissue	Not given	Not given	Not given				
Embryological	Right kidney normal. Left kidney 2 cm. lower than right showed acute changes due to infection from measles	Not given	Acute nephritis	None			
Abscesses in parenchyma. Pelvis dilated and filled with pus and concrements	Abscesses under capsule of right kidney. Pelvis dilated and filled with pus and concrements. Left kidney pelvis dilated and filled with thin pus and concrements	3 months	Progressive emaciation, constant fever, severe pains in back. Pus in urine	No	Cystitis following bullet wound. Suppurative pyelonephritis	Nephrectomy	Improvement for 2 months. Return of previous symptoms plus vomiting, diarrhœa, and hæmaturia. Urinary fistula opened. Death.
Dwarfed but perfect pyramids	Normal	As far back as patient can remember	Vaginal leakage of urine (6 years previous unsuccessful plastic operation for vesicovaginal fistula)	No	Supernumerary kidney with hydroureter opening into vagina	Nephrectomy	Cure.
Miniature. No structural evidence of its having functioned	Kidneys larger than normal	Not given	Not given	Not given			
None	None	None	Not given	Not given			
None	Smaller than kidney on right	Not given	Not given	Not given			
Infected hydronephros with dilatation of the ureter	Normal		Pain, nausea, vomiting, chills, and fever	Yes	Perinephritic abscess	Two nephrectomies	Cure.

Fig. 1. Right pyelogram made July 17, 1928, before the first operation. Note differences in the courses of the two ureters.

Fig. 2. Right pyelogram made July 26, 1928, after drainage of the supernumerary kidney.

largest number of cases reported occurred in the second decade of life, which would seem to justify the statement that this type of renal anomaly is prone to produce pathological changes in the kidney early in life.

SEX

A review of these 30 cases showed that 15 occurred in females, 14 in males, and in 1 case the sex was not mentioned, the incidence being about evenly divided between two sexes.

SIDE

With reference to the frequency with which either side is affected our series is so small that extra care should be exercised in interpreting the available data. However, in 17 the left side was affected, in 12 the right side, and in 1 case the location was not stated.

In the majority of the case reports the location was given rather accurately. In most of the cases the supernumerary kidney was found below the so-called normal kidney. The designation of the location varied from the simple term "below" to a definite anatomical description; for instance, Israel says partly above and behind the left kidney; Newman, close to the upper margin of the left kidney; Samuels, Kern, and Sacks, at the upper pole of the right kidney; Marine Hospital Report, in the hilum of the left kidney.

In the case which I previously reported, the supernumerary kidney was well below the so-called normal kidney, and in the case here reported it was behind the so-called normal kidney and extended much higher and much lower. The reason for this was that the kidney had been completely destroyed and only a very large hydronephrotic sac was present. The exact location of the supernumerary kidney is shown in the pyelogram (Fig. 4). The relationship in this case is somewhat analogous to that in the case reported by Fischer and Rosenloecher (Fig. 5).

COURSE OF THE URETER

In the case which I previously reported, the two ureters fused and formed a single ureter outside the bladder, so that repeated cystoscopic examinations showed two normally located ureteral openings. Exactly the same anatomical condition was found in the present case on cystoscopic examinations carefully carried out. As a rule, in this type of case only two ureteral orifices are found in the bladder, and because the

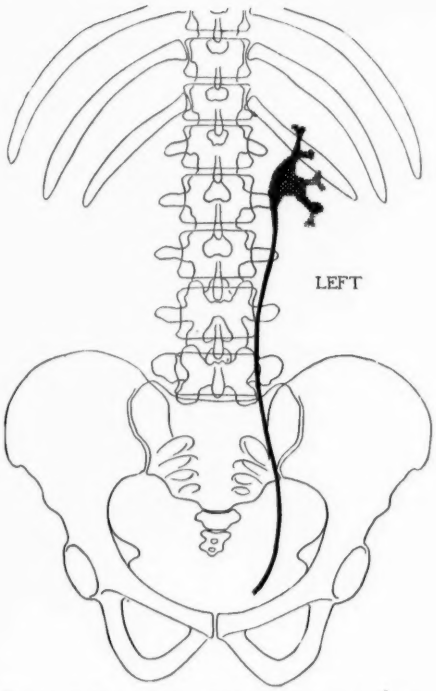

Fig. 3. Left pyelogram; kidney pelvis and ureter are normal.

cystoscopic picture is normal in this respect the reason this anomaly is not recognized is quite apparent.

The cases reported were studied to ascertain whether or not this course of the ureters was the rule, that is whether the two ureters on the side of the supernumerary kidney fused outside the bladder so that only two ureteral orifices were found in the bladder. Unfortunately, in some of the case reports this phase of the question was not discussed and data were not available on this point. Nevertheless, for the purpose of discussion the cases may be divided into four groups.

In Group 1 the course of the ureter could not be, or was not, traced so that information on this point was lacking.

In Group 2 the supernumerary ureter fused with the normal ureter. The fusion occurred anywhere along the course of the ureter with the result that two normal ureteral openings were found in the bladder on cystoscopic examination or at autopsy. Cases belonging to this group were reported by Dixon, Isaya, Kretschmer (2 cases), Linberg (Fig. 6), Maximovitsch, McArthur,

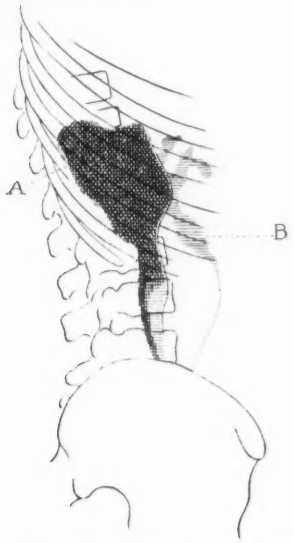

Fig. 4. Lateral view of the right side showing: (a) a large hydronephrotic kidney and (b) the pyelogram of the normal kidney on the corresponding side.

Newman, London and von Hansemann, and in the *London Medical Gazette*.

In Group 3 the ureters did not fuse outside the bladder so that there were three separate ureteral orifices. The location of the supernumerary ureteral orifice in the bladder varied greatly: above the right ureteral orifice (Fischer and Rosenloecher); in the fundus of the bladder (Hyrtl). In Clifford's case two of the three kidneys were rudimentary. Two ureters evidently had a normal termination in the bladder, and the third ended in a blind sac beneath the mucous membrane of the bladder between the ureteral and the urethral openings. Insertion of the supernumerary ureter may be vesical or extravesical, that is ectopic, as occurred in Camina's case in which the ureteral opening was found alongside the external urethral orifice; termination of the ureter in a cyst of the vagina was reported by

COURSE OF URETER

	Cases
Fused	11
Not fused	8
Not determined	7
Independent	1
Blind sac	1
Ureter lacking	1
Not clearly stated	1
Total	30

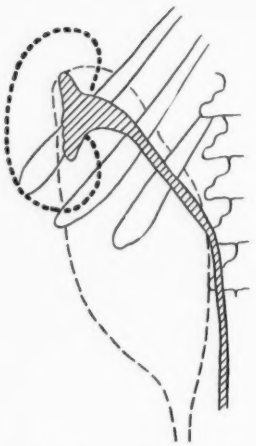

Fig. 5. Case of Fischer and Rosenloecher. Schematic representation of the pyonephrotic sac and the right kidney.

Israel, and in the anterior vaginal wall by Samuels, Kern, and Sacks.

In Group 4 we have the rudimentary ureter.

PATHOLOGY IN THE SUPERNUMERARY KIDNEY

An analysis of these 30 cases showed the presence of pathology in 19 instances. In a relatively large number of cases stone and infection were present, and they head the list as will be seen by referring to the table. Only 1 case of tumor was mentioned and, curiously enough, no case of renal tuberculosis is mentioned. Stones were present in 3 cases, and it is interesting enough to call attention to the fact that in two of them (McArthur and Kretschmer) stones were found not only in the supernumerary kidney but in the other kidney on the same side. In the case reported by Rutschinsky all three kidneys were the seat of stones and very severe infection. The supernumerary kidney was normal in 7 cases and in 4 cases no mention was made as to whether the supernumerary kidney was normal or not.

PATHOLOGY IN THE SUPERNUMERARY KIDNEY

	Cases
Stone	3
Rudimentary	3
Small but normal	3
Pyonephrosis	2
Ptosis	1
Lobulated	1
Papillary cystadenoma	1
Fibrosis with pyogenic infection	1
Cyst	1
Small and undeveloped	1
Hydronephrosis	1
Infected hydronephrosis	1
No pathology	7
Not stated	4
Total	30

PATHOLOGY IN THE OTHER KIDNEY ON THE SIDE AFFECTED

The other kidney on the side affected was normal in 14 cases and no mention was made of pathology in 6 cases, so that pathology was present in only 10 cases. Here, as in the supernumerary kidney, calculus was the condition most frequently found, having been reported in 4 of the 10 cases showing pathology.

PATHOLOGY IN OTHER KIDNEY ON THE SIDE AFFECTED

	Cases
Stone	4
Smaller than normal	2
Rudimentary	1
Ptosis	1
Pyonephrosis	1
Nephritic diagnosis	1
No pathology	14
Not stated	6
Total	30

In reviewing the pathology found both in the supernumerary kidney and in the other kidney, interesting figures are at hand. Of the 30 cases the supernumerary kidney showed pathology in 19 cases; 7 were normal, and no mention was made of the change in 4 cases. When one directs attention to the other kidney on the affected side, the number of cases in which pathology was found was 10, just about one-half. The kidney was normal in 14 cases and in 6 nothing was stated about the pathology. The largest single evidence of pathology was stone which occurred in 4 of these cases. Of course, it was during the operation for stone in some of these cases that the accessory kidney was found. In one case, that of Rutschinsky, calculi were present in all three kidneys. In the case which I previously reported there were stones in the supernumerary kidney and in the other kidney on the side affected.

The occurrence of rudimentary kidney is rare in this condition. Neckarsulmer coined the term "Beinieren" to designate, not the usual accessory kidney, but those which he stated should be placed in a separate class, third kidneys—distinct organs which appear only in rudimentary form and are of no functional importance. The author states that these organs may be called pararenal bodies in the same sense as the term parathyroid bodies is used to designate formations in the regions of the thyroid. Besides his case he calls

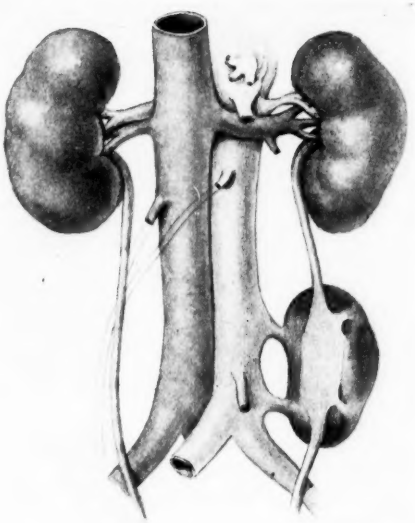

Fig. 6. Case of Linberg. Schematic representation of the position of supernumerary kidney.

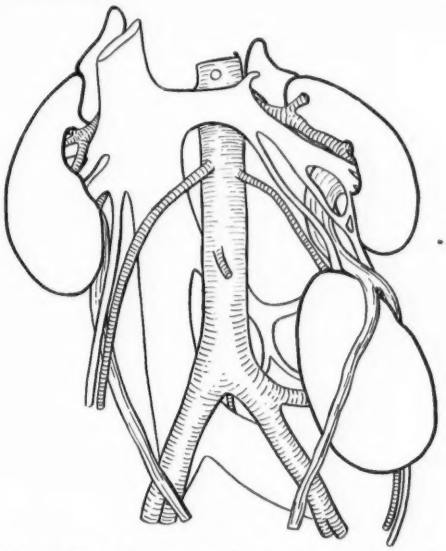

Fig. 7. Case of Dixon. Showing the occurrence of a third kidney in an adult male subject.

attention to the case reported by Schonberg and Palma. In conclusion, Neckarsulmer calls attention to the differences between these rudimentary organs and the fully developed third kidneys capable of functioning. While the latter show their independent character in most cases by being entirely separated from the normal kidney of that side, the rudimentary organs maintain a typographical connection with the kidney of that side. This fact, as well as their location on the upper pole in contradistinction to the separate kidneys which always lie toward the pelvis, is so constant that it might be called the rule.

In Clifford's case there were three rudimentary kidneys.

SYMPTOMS

Pain. The subjective symptoms vary, depending in part, of course, upon the underlying pathology. Pain was present in 16 cases and seems to have been the one outstanding constant symptom; but its description, duration, and localization varied greatly, being described as epigastric, abdominal, renal colic, and pain in the left side of the abdomen. In Calabresse's case the symptoms were of 2 weeks' duration, whereas in Isaya's case they were present for 21 years. Among other symptoms noted were nausea, vomiting, frequency of urination, strangury, and pain in bladder.

There was nothing characteristic in the symptoms mentioned by patients to allow one to establish a diagnosis of supernumerary kidney; often the symptoms were suggestive of a kidney lesion, but beyond this one could not venture a diagnosis without resorting to special methods.

PALPABLE TUMOR

Palpable abdominal tumor was found in 14 cases. No tumor was present in 6 cases, and no mention was made on this point in 9 cases. In 1 case a swelling was palpable, but, as subsequent events proved, the palpable swelling was due to a dermoid cyst and hence had nothing to do with the supernumerary kidney. In the case which I previously reported, the supernumerary kidney was very small, hence, it was not palpable. In the case herewith reported, a palpable tumor was present due to a very large hydronephrosis that extended from under the arch of the ribs into the right iliac fossa. That the palpable tumor in this case was due to a supernumerary kidney did not dawn upon us.

DIAGNOSIS

A pre-operative diagnosis was not made in many of these cases. This is easy to understand in the older cases before the present accurate methods of diagnosis were available. A pre-operative diagnosis was made by Israel and by Samuels, Kern, and Sacks.

In the group in which the kidney and its ureter are rudimentary, pre-operative diagnosis is almost

never, if ever, made. The difficulties in establishing a diagnosis in this rare type of case is, of course, self-evident. Such cases in the literature were all discovered at autopsy. (Clifford, Mangeti, and Marine Hospital Report.)

The group in which the two ureters on one side fuse outside the bladder to terminate as one ureter in a normally placed ureteral hillock presents difficulties in diagnosis in that the cystoscopic picture is that of a normal bladder with two ureteral orifices. If a pyelogram is made with the catheter low down, a picture of both kidneys may be obtained and a positive diagnosis made.

In the case herewith reported the pre-operative diagnosis rested between perinephritic abscess and carbuncle of the kidney. The diagnosis of three kidneys was made at the time of the first operation and was verified by an injection into the drainage tube in the loin with a simultaneous pyelogram, which showed the pelvis of the normal kidney as well as the large hydronephrotic sac of the supernumerary kidney.

In the third group in which the ureters do not fuse and the three ureters are seen in the bladder, or in which they terminate extravesically, a clue to the diagnosis is at hand and may be verified by pyelograms. In this group, of course, it is necessary to differentiate between supernumerary kidney and double kidney with an accessory ureter.

TREATMENT

Treatment must, of course, vary, depending upon the location of the disease and especially on the type of disease. If, for example, the lesion is in the kidney on the same side, then the supernumerary kidney may be left *in siu*. When the lesion occurs in the kidney on the same side, there are good grounds to suspect the presence of a supernumerary kidney. This happened in several of the reported cases.

The following surgical operations have been performed.

TABLE OF OPERATIONS

	Cases
No operation	11
Nephrectomy	10
Nephrectomy of both the supernumerary kidney and the kidney on the corresponding side	2
Nephrotomy	1
Pyelotomy	1
Nephropexy of both left kidneys	1
Nephropexy and nephrectomy	1
Exploratory only	1
Found during operation for ptosed colon	1
Removal of tumor	1
Total	30

SUMMARY

1. The primary lesion in the case reported in this paper was a very large infected hydronephrosis.

2. There was no communication between the two kidneys; each had a separate blood supply and a separate ureter that fused below the brim of the pelvis and outside the bladder.

3. The two kidneys on the right side were removed.

4. The total number of cases reported to date including this case is 30.

REFERENCES

1. Blasius, G. Observationes medicæ rariores. Abrahamum Wolfgang, 1677. Part iv, Morbos in numero continens, observation, xvi, p. 58.
2. Calabrese. Rein surnumeraire constate pendant la vie. Ann. d. mal. d. org. génito-urin., Par., 1908, ii, 1841-1847.
3. Caminã, Ramon. La concentracion maxima on casos de rinones suplementarios. Med. Ibera, Madrid, 1920, xiii, 216–219.
4. Cheyne, W. Watson. A case of movable third kidney. Lancet, Lond., 1899, i, 215.
5. Clifford, A. B. Two cases of abnormal kidney. United States Nav. M. Bull., 1908, ii, 37.
6. Cobb, F., and Giddings, H. G. Supernumerary kidney, subject of cystadenoma. Ann. Surg., 1911, liii, 367–372.
7. Depage. Un cas de rein surnumeraire pris pour un ganglion tuberculeux du mesentere. Extirpation. Rein mobile a droite. Nephropexie. Guerison. J. de méd. de chir. et de pharmacol. de Bruxelles, 1893, xcv, 167–170.
8. Dixon, A. F. Supernumerary kidney; the occurrence of three kidneys in an adult male subject. J. Anat. & Physiol., 1910–11, xlv, 117–121.
9. Fischer, K., and Rosenloecher, K. Ueber einen Fall von dritter Niere mit selbstaendigem Harnleiter. Ztschr. f. urol. Chir., 1925, xvii, 61–66.
10. Gayet. Rein surnumeraire. Lyon méd., 1923, cxxxii, 1061.
11. Holmes H. I. A third kidney with inflammatory changes. Austral. M. J., Melbourne, 1911, i, 116.
12. Hyrtl. Ein wahrer Ren tertius. Osterr. med. Jahrb., Wein, 1841, 965–967.
13. Isaja, A. Rene soprannumerario constatato durante la vita. Ann. del r. Ist. di chil., chir. di Roma, 1912, iv, 369–378.
14. Israel, I. Diagnose und Operation einer ueberzaehligen pyonephrotischen Niere. Berl. klin. Wchnschr., 1918, lv, 1081.
15. Kretschmer, H. L. Supernumerary kidney. J. Am. M. Ass., Chicago, 1915, lxv, 1447–1448.
16. Kuksinskaja. Cyste einer ueberzaehligen Niere. Ztschr. f. Urol., 1927, xxi, 342–346.
17. Lebedeff, A. I. Case of movable kidney (accessory) simulating a dermoid cyst of an ovary. Vrach. Leningrad, 1897, no. 18, 539.
18. Linberg, B. E. Zur Frage der Nierenanomalien. Ztschr. f. urol. Chir., 1924, xv, 315–321.
19. Mangeti, Joh. Jacobi. Theatrum anatomicum. Geneva: Cramer & Perachon, 1717. Eustachii tabule anatomicae. 2d ed.

20. MAXIMOVITSCH, A. S. Case of accessory kidney. Ztschr. f. Urol., 1927, xxi, 801–802.
21. MCARTHUR, L. L. Some renal anomalies. St. Paul M. J., 1908, x, 440–445.
22. MUNRO, J. C., and GODDARD, S. W. Pyelonephrosis of a supernumerary kidney. Am. J. M. Sc., 1907, cxxxiv, 398–403.
23. NEWMAN, D. Malformation of the kidney and displacements without mobility, with illustrative cases and specimens. Tr. Clin. Soc., Lond., 1898, xxxi, 118–157.
24. NECKARSULMER, K. Ueber Beinieren. Berl. klin. Wchnschr., 1914, li, 1641–1643.
25. RUTSCHINSKY, B. P. Multiple deformities of the kidneys. Vrach. Gaz., Leningrad, 1907, no. 22, 620–621.
26. SAMUELS, A., KERN H., and SACHS, L. Supernumerary kidney with ureter opening into vagina. Surg., Gynec. & Obst., 1922, xxxv, 599–603.
27. Supernumerary kidney. Annual Report of the Supervising Surg. Gen. Marine Hospital of the U. S., 1884–1885, 148–150.
28. Supernumerary kidney. London M. Gaz., 1838, ii, 127.
29. VON HANSEMANN. Ueberzaehlige Nieren. Berl. klin. Wchnschr., 1897, p. 81.

A LARGE CYSTIC MYOMA SITUATED IN THE LEFT BROAD LIGAMENT AND ALMOST COMPLETELY SEPARATED FROM THE UTERUS

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CYSTIC myomata, similar to the one to be described, are exceptionally rare. Kelly and Cullen, in a review of over 1,600 myomata seen by them, did not encounter a similar case, and in going through the literature I found only 4 such cases. Janicot and A. L. Smith described large myomatous tumors with a distinct connection still existing between the growth and the uterus. Eden and A. J. Smith found only a few strands of tissue between the two structures.

The similarity between the various growths described was marked. All the myomata were within the broad ligaments. The tumors were large and two (4, 5) had dissected the peritoneum free from the posterior surface of the uterus right across into the broad ligament of the opposite side. It is of interest to note that in all 4 cases the growth had arisen on the left side. In every case the intraligamentary position of the myoma simplified removal, because the tumor could be shelled out with little bleeding and without danger to the ureter.

The tumors consisted largely of multiple cysts together with small masses of myomatous and fibrous tissue. Even the densest parts contained small cysts, some being microscopic in size. All stages of change from muscle and fibrous tissue through hyaline degeneration to the clear fluid of the cysts were noted. Janicot claimed that one cavity was lined with endothelium, but no one else found any sign of a lining membrane in the cavities. In each case there still remained some connection between the myoma and the myometrium. In spite of this both Eden and A. J. Smith made the definite assertion that the myomata had sprung from smooth muscle fibers normally present in the broad ligaments.

In Eden's case death occurred 33 days after operation from some ill defined pulmonary condition. The other patients made uneventful recoveries.

Mrs. S. B., aged 43, white, consulted Dr. Thomas S. Cullen on September 19, 1928. Her periods had been regular and painless. She had had one child. For 5 years she had had a "disagreeable feeling" in the left lower abdomen. On examination, some tenderness was noted in the appendix region, and the patient complained of a "gnawing sensation" in the left lower abdomen. The outlet was normal, the cervix back, and the body of the uterus normal in size. The left side of the pelvis was filled with what appeared to be a cystic tumor. This also occupied the lower portion of the left side of the abdomen.

Operation, Women's Hospital, September 24, 1928. As soon as Dr. Cullen had opened the abdomen, it was perfectly clear that there was an intraligamentary tumor on the left side, as there were two series of blood vessels. The broad ligament peritoneum was split and the tumor shelled out. There was very little bleeding and a large hole was left in the broad ligament. There was some oozing down near the left uterine artery. It was necessary to dissect out the left ureter after which the four or five bleeding points were readily controlled. The broad ligament was then closed. There was a myoma about 2 centimeters in diameter far down on the posterior surface of the uterus. It was not disturbed. Both tubes and ovaries were normal. The appendix was looped on itself and adherent. It had evidently given rise to considerable trouble. It was removed. The abdomen was closed without drainage.

Following operation the patient developed a phlebitis in the left leg. She had had some trouble with the outer side of this leg before operation. She made a satisfactory recovery.

Fig. 1. Gyn. Path. No. 33838. Myoma of the broad ligament. For its pedicle, see Figure 2. For its appearance on section, see Figure 3.

Fig. 2. Gyn. Path. No. 33838. Myoma of the broad ligament. The pedicle is shown in the center of the picture. All that was necessary was to tie a few small vessels. The tumor had hardly any connection with the uterus.

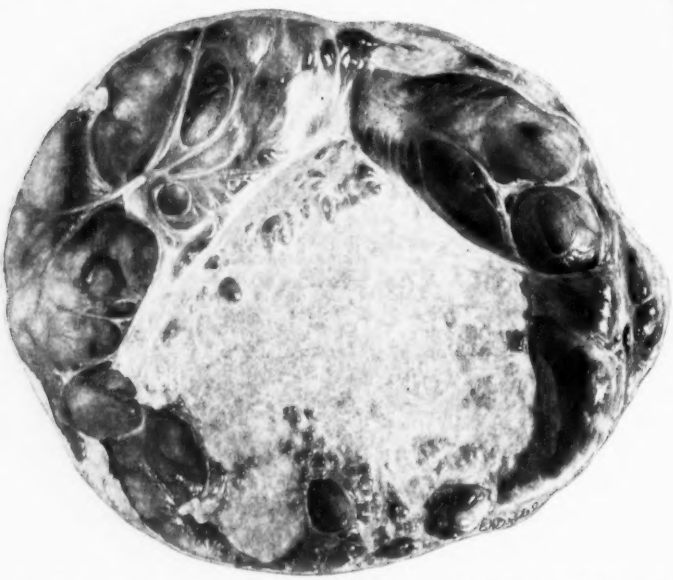

Fig. 3. Gyn. Path. 33838. A large cystic myoma which lay in the left broad ligament and had only the faintest connection with the uterus. For the appearance of the outer surface of the tumor, see Figures 1 and 2. In the center is a core of typical myomatous tissue. Passing off from this toward the periphery are numerous trabeculæ. The cystic spaces had no epithelial or endothelial lining. The cavities were filled with serous fluid.

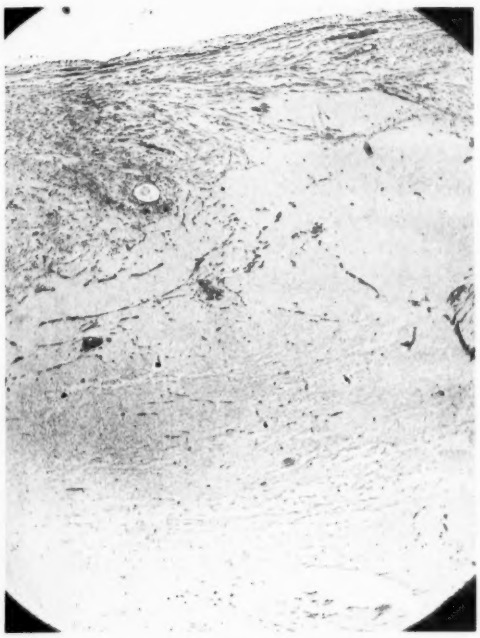

Fig. 4. Gyn. Path. No. 33838. Myomatous tissue undergoing liquefaction. In the upper part of the picture myomatous tissue is clearly seen. As we pass downward, the muscle cells rapidly diminish and then totally disappear. In the lower part of the field liquefaction has taken place.

Examination of the patient on November 27, 1928, showed normal pelvic structures. There was not the slightest thickening in the left broad ligament.

Pathological report. Gyn.-Path. No. 33838. The specimen consists of a partly solid, partly cystic tumor, 16 by 14 by 8 centimeters (Fig. 1). It is uniformly grayish in color, is devoid of peritoneum, and covering its surface are a number of fan-like thin adhesions. At one point is a small pedicle, about 1.5 centimeters in diameter (Fig. 2). This contains two or three small blood vessels.

On section, one finds a central core resembling myomatous tissue, and, passing from this to the outer surface of the tumor, are trabeculæ (Fig. 3). The greater portion of the tumor, in the outlying areas, is cystic. The line of demarcation between the cystic areas and the solid portion is sharply defined. The cystic cavities contain yellow, serous-like fluid.

Sections through the central core show that it is a myoma (Fig. 4). There is, however, a great deal of hyaline transformation, so that just here and there is a muscle fiber visible. At other points, the hyaline material has liquefied or has entirely disappeared, leaving a fibrillated tissue (Fig. 5). In some places, hyaline transformation has occurred in clumps, squeezing the remaining muscle fibers and producing a most unusual picture. The remaining fibers look like channels of cells running in all directions. The cystic cavities, noted in the outlying portions of the tumor, have no cellular lining. They are due to a breaking down and liquefaction of the hyaline myomatous tissue.

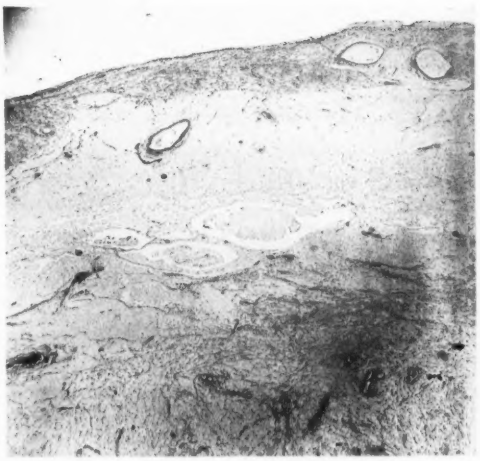

Fig. 5. Gyn. Path. No. 33838. A degenerating myoma. In the upper part of the section is myomatous tissue. In the central portion is an area of liquefaction. In the lower half of the section are some muscle fibers, but the major portion of the picture consists of delicately fibrillated tissue devoid of nuclei.

We have, in this case, a partly cystic myoma which has, in large measure, become separated from the uterus, and which is lying in the left broad ligament. There is not the slightest evidence of malignancy.

I want to thank Miss Elizabeth Broedel for her fine illustrations, Mr. Hermann Becker for his clear photomicrographs, Dr. Cullen for asking me to report this unusual case.

REFERENCES

1. Eden, T. W. A case of intraligamentous fibrocystic tumor of the uterus, weighing about 30 pounds, removed by enucleation and subtotal hysterectomy. Tr. Obst. Soc. Lond., 1907, xlviii, 264–271.
2. Janicot, R. Pr'sentation d'une tumeur fibro-kystique de l'ut'rus incluse dans le ligament large, et reliée au fond de l'uterus par un p'dicule. Bull. Soc. d'anat. et physiol. de Bordeaux, 1901, xxii, 145-148.
3. Kelly and Cullen. Myoma of the Uterus. Philadelphia: W. B. Saunders and Company, 1909.
4. Smith, A. J. A bilateral intraligamentous fibromyoma showing extensive mucoid degeneration. Brit. Gynæc. J., 1902, xviii, 121–128; Med. Press and Circ., 1902, lxxviii, 540.
5. Smith A. L. Case of fibrocystic tumor of the uterus filling left broad ligament; removal, recovery. Dominion M. Month., 1902, xix, 324–325.

OSTEOCHONDRITIS OF THE SYMPHYSIS PUBIS

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OSTEOCHONDRITIS of the symphysis pubis is probably not an extremely rare condition, but it is one about which there is but scant information in the literature. Because of this lack of literature on the subject, and because of the fact that in at least 3 of these 4 cases the diagnosis was overlooked for a long time by competent surgeons, we feel that it is worth while to bring this condition to the notice of the profession. The predominance of urinary symptoms in such patients, or the occurrence of osteochondritis as a complication of bladder surgery, usually brings such cases to the urologist who is not particularly interested in bone pathology. This fact probably accounts, in part, for the lack of literature on the subject. Three of the cases were seen by us on the genito-urinary service of the Massachusetts General Hospital. The fourth was a private case of Dr. Arthur Chute, of Boston, to whom we are greatly indebted for permission to include it in this report.

Tuberculosis of the symphysis pubis and osteomyelitis of other bones of the pelvis are not infrequently referred to in the literature. Cabot refers to infection of the symphysis pubis as a possible complication of suprapubic prostatectomy. Legueu and Rochet report the occurrence of an abscess behind the pubic bone as a complication of suprapubic prostatectomy, causing in one case erosion of the pubic bone. This case they thought was due to direct infection from the urine. The only extensive works on the subject which we have been able to find are two articles by Beer. In all, he reports that he has seen 12 or more cases of periosteitis and osteitis of the symphysis pubis following suprapubic cystotomy. He believes that the condition is caused either by injury to the periosteum from traction on the recti muscles, or by pressure from the suprapubic drainage tube. The symptoms develop 3 or 4 weeks after operation. There is usually pain when patient moves and walks, due to the pull of the muscles on the diseased periosteum. On examination there is slight tenderness over the pubis. The X-ray picture of the pubis may be normal, or may show various degrees of involvement of the pubic bone, from slight changes in outline to a marked destruction. As the process extends, it may involve the descending ramus of the pubis. In all cases, Beer found that the condition cleared up after simple local treatment, such as the application of heat.

Two of our cases were similar to the ones reported by Beer, in that they followed suprapubic prostatectomy. They differed, however, in that they did not go on to a spontaneous cure. The other two did not follow an operation on the bladder. In fact, all 4 cases differed from one another in many respects. Each case, however, showed signs and symptoms of a septic process involving the symphysis, which was proved to exist either at operation or at autopsy. Likewise the condition showed in every case marked chronicity, which tended to obscure the diagnosis, as did also the lack of definite X-ray findings in 2 of the cases. In these 2 cases the X-rays were at first interpreted as being normal by an expert roentgenologist. The interpretation of an infectious process in this bone is very difficult, because of the wide anatomical variations in the outline of this joint. In every case the infectious process was due, so far as we have been able to determine, to a pyogenic organism and not to tuberculosis.

The first case (M. G. H. 271355) of osteochondritis of the symphysis pubis which we saw was in a man of 69 years. He entered the Massachusetts General Hospital on January 3, 1928, complaining of the typical symptoms of benign prostatic hypertrophy. Two days after entry, a suprapubic cystotomy was done under local anæsthesia. A large rubber tube was inserted into the bladder for drainage, and an empty Miller wick was placed in the prevesical space. The incision was carried down unusually close to the pubic bone, and the drainage was from the lower angle of the incision, but so far as was known, the periosteum of the pubis was not injured. Three weeks later, a second stage suprapubic prostatectomy was done under ethylene anæsthesia. Fifteen days later the suprapubic wound was healed, the patient was voiding normally, and was well enough to go home.

Two months after discharge the patient entered the emergency ward of the hospital. At this time he stated that, since his discharge from the hospital, his legs had felt weak and he had had a drawing sensation in the region of the pubis. Ten days before entry this sensation had changed to a severe pain in the lower abdomen, which radiated down both legs. This pain was aggravated by motion and had become so severe that the patient was unable to walk. At this time he had practically no urinary symptoms.

Physical examination showed a well-healed suprapubic scar, at the lower end of which an indefinite mass could be felt in the prevesical space. This was tender, about the size of a lemon, and firmly attached to the pubic bone. The skin over this area was slightly red. Any movement of the legs was painful, but compression of the crests of the ilium produced no discomfort. The urine was grossly clear, but contained white blood cells on microscopic examination.

Fig. 1. Roentgenogram of the first case reported. This was taken just before operation and shows only slight irregularity of the outline of the pubic symphysis which is within normal limits. At operation the cartilage was found destroyed but the bone did not appear to be involved.

Fig. 2. Roentgenogram of the third case. The symphysis pubis is irregular in outline and shows disalignment with the sacrum. Areas of decreased density in the left pubic bone are not well shown in the reproduction.

The white blood count was repeated several times and was normal except on one occasion. The temperature varied between 99.5 and 98.0 degrees. An X-ray taken at this time showed slight irregularity of the outline of the pubic bone adjacent to the symphysis, but was considered by the roentgenologist to be within normal limits. The patient was observed for 2 weeks in the hospital; the local signs subsided, his symptoms improved markedly, and he was discharged without any diagnosis having been made.

On May 9, 1928, nearly 4 months after his operation, the patient returned again to the hospital. At this time his symptoms were the same as at his last entry, only not so severe. There was tenderness over the pubic bone, but no mass could be felt. He had lost some weight, but his general condition was good. The temperature and white blood count were normal, but the urine was grossly infected. Cystoscopy showed an area of granulation tissue on the roof of the bladder in the region of the old incision. This area was about 1 centimeter in diameter, and a small stream of pus could be seen entering the bladder from beneath it. Accordingly a diagnosis of prevesical abscess draining into the bladder was made. The X-ray at this time (Fig. 1) was the same as 2 months before, and was again reported as negative.

On May 17 operation was performed. The old scar was excised, the peritoneum was freed from the bladder, and the bladder was opened. The bladder was adherent to the pubic bone, from which it had to be separated by sharp dissection. After this, the bladder was closed. The incision was then carried down on to the pubic bone through a mass of indurated tissue. Some yellow pus escaped, which, on culture, grew a Grahm negative bacillus. The cartilage of the symphysis was found completely destroyed. Although the rami of the pubis were thus completely separated from each other, they did not appear involved in the process in any way. Some tissue in this region was removed for pathological examination, and report of chronic inflammatory tissue was returned by the pathologist. Two cigarette drains were inserted and the wound closed in the usual manner.

Three weeks later the patient was discharged, relieved, but with a small draining suprapubic sinus. This sinus persisted for 3 weeks after discharge, and then healed of its own accord. He continued to have pain in legs and region of the pubis on walking for several weeks after the operation. This has, however, slowly disappeared, so that at the present time, 11 months after operation, he has no pain except occasionally after very severe exertion. His general condition is good and he has gained weight.

This case is very interesting and shows the typical symptoms of an infection involving the symphysis. Tenderness and pain in the region of the symphysis and pain on motion of the legs are the outstanding signs and symptoms. We believe that the infection must have resulted from the original operation. It is more likely that the injury to the symphysis was caused either directly at the time of operation, or by placing the suprapubic drainage tube too near the pubis, than that it was caused by undue retraction on the recti muscles, as suggested by Beer. It could, we believe, have been avoided by a slightly higher incision and by bringing the suprapubic tube out farther from the pubis. The absence of a definite abscess in the space of Retzius made us think that the symphysis was primarily infected and that the osteochondritis did not develop secondarily to a prevesical abscess, although we cannot definitely prove this contention. The marked chronicity of the condition, and lack of signs of active infection, were suggestive of tuberculosis, but this diagnosis has been ruled out by the culture, the biopsy, and the subsequent course of the disease. The lack of X-ray evidence of the condition was, of course, due to the fact that the infection involved only the cartilage of the joint. Although Beer states that this rather unusual complication of prostatectomy subsides without operation, this case continued for 4 months, finally ruptured into the bladder, and was cured only by operation.

The second case (J. B. Q.) had a suprapubic cystotomy on December 20, 1927, and a suprapubic enucleation of the benign prostate on January 9, 1928. Following the second operation, he began to run a septic temperature and to show signs of general sepsis. The usual laboratory tests were done, and the patient was seen by various consultants, but they were unable to determine the location of the sepsis. In 3 weeks the suprapubic incision had healed, but the patient continued to go down hill and to run a septic temperature without definite localizing signs. About 7 weeks after the second operation, tenderness was noted in the region of the pubis and the suprapubic wound broke open and drained pus. Three weeks after that, an X-ray of the pelvis showed marked osteomyelitis of the symphysis pubis. Three months after the first operation, the patient died.

At autopsy there was found an abscess in the space of Retzius which had eroded the pubic bone and completely destroyed the cartilage of the symphysis pubis. In addition to this, however, there was a bacterial endocarditis, a low-grade peritonitis, infarcts of the spleen, abscesses of both kidneys, thrombosis of the pelvis and left renal veins, and streptothricosis of the lung. Cultures from the abscess, the heart, and peritoneal cavity 4 hours after death, grew bacillus coli.

In this case, although the abscess in the prevesical space associated with the osteochondritis may have been the primary source of infection, it seems much more logical to assume that the osteochondritis was secondary to the general septicæmia.

The third case (M. G. H. 288974), a young Canadian of 25 years, entered the hospital on January 31, 1928. He complained of pain in the region of the pubis of 7 months' duration. In his work he had been carrying heavy boxes, supporting them partly on his lower abdomen. The pain had been persistent as a dull ache, often radiating down the left leg and sometimes also down the right. Working always made the pain worse so that recently he had had to stop work altogether. Associated with this he had marked frequency, every half hour by day, but no nocturia. He sometimes had marked urgency, making the pain worse, at which times he would be somewhat relieved by urination. Six months previous he had noticed a small lump in the left inguinal region, which had since disappeared. Aside from these symptoms, his history was unimportant.

Physical examination was essentially negative except for some tenderness just behind the pubis. The prostate was large, soft, and very tender, but secretion obtained from it did not show pus. A complete urological examination, including pyelograms, was negative. The X-ray of the pubic bone showed some irregularity in the pubic symphysis, and also some asymmetry and disalignment of the symphysis with the sacrum. It was felt by the radiologist that the irregularity was probably only an anatomical variation and that the asymmetry was due to the position of the patient. The patient was observed in the hospital for 2 weeks. During this time his temperature on occasions varied between 97.5 and 99.5 degrees. His white blood count was normal. His symptoms all disappeared, and he was discharged without any diagnosis having been made.

Ten months later he returned to the hospital and reported that he had been well for 9 months. One month previous all his old symptoms had returned. He had pain over the pubis and in the left inguinal region. In addition, he had frequency and marked dysuria.

Examination showed marked tenderness and spasm above the pubis and many large tender glands in the left inguinal and femoral region. The prostate was large and soft, and above it there was an indefinite, soft, tender mass. The urine was negative. On entry the temperature was normal, but it soon began to rise. Four days later it reached 102 degrees. At this time the white blood count was 21,000, and a very definite tender mass could be palpated bimanually in the region of the bladder. An X-ray (Fig. 2) plate showed the same condition as in the one taken 10 months before, except that the irregularity of the articular surface of the pubis was more marked, and the ascending ramus of the left pubis showed three areas of decreased density associated with some increase in density of the descending ramus.

Operation was performed on the following day. A low mid-line incision was carried down between the recti muscles, opening into a large abscess in the prevesical space. A culture of pus from the abscess grew staphylococcus aureus. The abscess cavity extended around the front and sides of the bladder, which was not opened, and beneath the pubis Cigarette drains were inserted. The patient made an uneventful recovery. After operation the prostate did not feel abnormal, but the vesicles were large and tender, and the secretion obtained on massage showed twenty white blood cells. He was discharged 5 weeks after operation with a small draining suprapubic sinus.

Four months after operation the patient reports that this sinus has never completely healed and that it drains a little pus from time to time. He has, however, had no pain or urinary symptoms. He has gained weight, feels well, and is working regularly.

This we consider a very interesting case, and as we know of no other report of primary non-tuberculous osteochondritis of the symphysis pubis, we hesitate to discuss its etiology. The infection of the pubic bone may, of course, have come from the prostate and vesicles, but the fact that no pus was obtained in the prostatic secretion when the patient was first seen, and the fact that cultures from the prevesical abscess grew staphylococcus make us think that this infection began primarily in the pubic bone. The urinary symptoms were, we believe, due only to the presence of a septic process outside the bladder. The marked chronicity of the condition and the small amount of change in the X-ray plate made the diagnosis difficult. We feel, however, upon looking back in the light of our present knowledge, that we should have been able to make the diagnosis when the patient was first seen, and that operation and drainage at that time should have been carried out. Like all forms of osteomyelitis, the treatment has not been very satisfactory, and a draining sinus has persisted. This might, perhaps, have been avoided if the bone had been curetted at the time of operation.

The fourth case (M. G. H. 295825) came to the hospital complaining of a urinary fistula. Thirteen years previous he had had an external urethrotomy for stricture. Eight years ago he had had a repair of bilateral inguinal hernia in another hospital. At this time the right incision became septic and drained pus for 5 years, healing only 3 years ago. Seven months ago a swelling developed at the right of the

old sinus. This broke down and began to drain pus and urine, which it continued to do until entry. Nine weeks ago a suprapubic cystotomy with curettage of the sinus was performed, also in another hospital. He left this hospital 1 week ago.

The important findings on physical examination, from the point of view of this report, were as follows: an open suprapubic wound draining pus, marked inflammation of the skin, tenderness over the pubis, and a sinus in the right groin in the region of the old hernia scar. This sinus extended downward and backward toward the prostate. A small amount of pus and urine drained from it continually, and upon urination about one-third of the urine passed through the sinus. The prostate was not abnormal, and a No. 28 sound could easily be passed into the bladder. Cystoscopic examination showed that the bladder was normal and that the sinus entered into the prostatic urethra.

An X-ray (Fig. 3) picture revealed apparent fusion of the symphysis pubis and considerable bone reaction around the symphysis without bone destruction. The changes were characteristic of an infectious process beginning in the symphysis pubis. Although this patient had been carefully studied and treated for this urinary fistula during the previous 2 months by one of the leading surgeons of the country, the presence of the associated infection of the symphysis pubis had not been recognized. Several attempts were made to drain the bladder with a urethral catheter, but the catheter was not tolerated. Finally, the bladder was drained through an external urethrotomy incision, and the sinus was curetted and partially excised. At this time rough, bare bone could be felt on the posterior aspect of the pubis.

The infection of the symphysis pubis in this case may have resulted originally from a prostatic abscess, or it may have resulted from infection at the time of the hernia operation. The latter explanation seems to be the more logical, as it explains why a sinus persisted at the site of the hernia scar for 5 years following operation. The urinary fistula, it seems, might have developed as a result of a prevesical abscess coming from the osteochondritis of the pubis and rupturing into the urethra, as it ruptured into the bladder in the first case. The fusion of symphysis pubis explains the lack of pain on motion which is usually an outstanding symptom. The marked chronicity suggests tuberculosis, but we have been unable either definitely to prove or disprove this suggestion, in this case. The X-ray findings are not, however, suggestive of tuberculosis. At the time of discharge, 3 weeks after operation, the patient was voiding normally and all wounds had healed except a sinus in the right groin which was draining a small amount of pus. Since discharge the patient has unfortunately been lost track of, so that we do not know his present condition. It seems unlikely, however, that the condition can be cured without completely eradicating the infection in the pubic bone.

SUMMARY AND CONCLUSION

As we have seen four cases of osteochondritis of the symphysis pubis in a year, and as Beer reports seeing a case or more a year for 12 years, we believe that the condition is much more common than one would be led to believe from a review of the literature. The frequent lack of definite X-ray findings, marked chronicity of the condition, and the fact that many cases get well without operation may result in the diagnosis being missed. However, if the possibility of this condition is considered when the case is being examined, this will probably not occur. The most common etiology is infection as a result of suprapubic cystotomy.

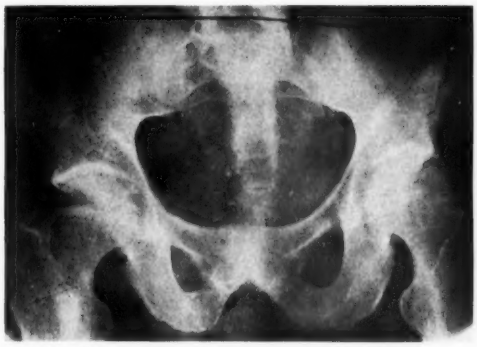

Fig. 3. Roentgenogram of the fourth case showing marked proliferation of bone completely obliterating the pubic symphysis. This is a characteristic picture of an old infectious process in the bone.

We have reported 4 cases. One case followed directly from a simple suprapubic prostatectomy; a second one came either from this same cause or as a result of a general septic process; a third case was apparently primary in nature; and a fourth case probably followed an operation for inguinal hernia.

Although Beer found that his cases (all of which followed suprapubic cystotomy) got well without operation, our cases have all persisted for a long time and were relieved only by operation. In 3 of our cases, the operations were moderately successful. The fourth case died of septicæmia.

The most constant symptom was pain in the region of the pubis, radiating down the legs and made worse by motion. One case showed marked urinary symptoms. Definite tenderness is present over the pubis in every case; other signs of a local inflammatory process in the region of the symphysis may or may not be present. As the cartilage seems to be first involved, the X-ray findings may not be definite. They are particularly hard to interpret as the symphysis pubis shows wide anatomical variations in the normal case.

REFERENCES

1. Beer, E. Periosteitis of the symphysis and descending rami of the pubis following suprapubic operations. Internat. J. Med. & Surg., 1924, xxxvii, 224–225 (May, 1924).
2. Ibid. J. Urol., 1928, xx, 185–196.
3. Cabot, H. Modern Urology. Philadelphia: Lea and Febiger, 1918.
4. Legueu and Rochet. Pelvis cellulitis after cystostomies and suprapubic prostatectomies. J. d'Urol., 1923, January, 1–11.

FRACTURES OF THE CARPUS

A Study of Twenty-Five Unselected Cases[1]

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THE persistent pain and limitation of function following carpal damage is distressing to the patient and annoying to the surgeon. The discomfort is not alone associated with displaced fractures and dislocations but even with fissured fractures. To ascertain the reasons for these discomforts, a study of 25 consecutive cases of carpal injury treated at the New York Post-Graduate Hospital was undertaken. To begin with, a superficial review of the anatomy is in order.

ANATOMY

Each carpal bone consists of "cancellous tissue enclosed in a layer of compact bone" (Spitzka). The articular surfaces are smooth and covered with cartilage and synovial membrane, the other surfaces are rough for the attachment of ligaments and muscles. "The blood supply of the various bones is quite scant" (Speed).

The scaphoid, semilunar, and sometimes the cuneiform articulate with the lower end of the radius to form the wrist joint. The wrist joint articulation is truly condyloid and allows of all the movements of such a joint, except rotation, which motion is taken care of by supination and pronation of the forearm. Circumduction at the wrist joint is limited by the styloids of the radius and ulna.

The scaphoid lies between the lower end of the radius and the os magnum, acting as a buffer between the latter two bones. The os magnum fits like a wedge into the hollow of the scaphoid, which, in turn, fits into the articular surface of the radius. This is particularly noted when the hand is in radial flexion. The tubercle of the scaphoid is extra-articular. The dorsal ledge of the lower articular surface of the radius hangs lower than does the palmar. When the hand is in complete dorsiflexion (hyperextension) the lower end of the radius impinges upon the os magnum, while the semilunar lies immediately beneath the flexor tendons. The joint is crossed by tendons on all sides. Occasionally a few fibers of the abductor pollicis muscle are attached to the tubercle of the scaphoid. Some of the other carpal—pisiform and trapezium—also have muscle attachments but from the standpoint of this discussion, are of no great interest.

MECHANISM OF INJURIES

When the hand is in dorsal and radial flexion, which is the position assumed in falls upon the outstretched hand, the scaphoid is in direct contact with the radius and lies between that bone and the os magnum as heretofore stated, so that a violence starting in the hand is carried through the os magnum to the scaphoid and thence to the radius. If the radius does not give (Colles' or kindred fracture), the scaphoid cannot escape and, receiving the full force of the violence with the wedge-shaped os magnum jammed sharply against it, breaks. If the force continues, the anterior ligament gives with a resulting dislocation of the semilunar.

If the hand is in dorsal and ulnar flexion, the violence is transmitted through the semilunar against the anterior ligament which tears, allowing the semilunar to escape through the rent in the capsule and a dislocation of the semilunar occurs. This last type of injury is not common without a concomitant fracture or dislocation of one of the other carpals or fracture of the radius. Destot states that isolated displacement of the semilunar without luxation or fracture of the scaphoid is never seen. The most common cause for carpal damage in our series was indirect violence, usually a fall from a height. When direct violence was the causative agent (1 case) a crushing injury of the soft structures was the result and associated therewith was a wound of

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Fig. 1. Air-injected wrist joint. Cadaveric specimen showing fracture of scaphoid. Triangular cartilage. (Courtesy of Dr. W. W. Lasher.)

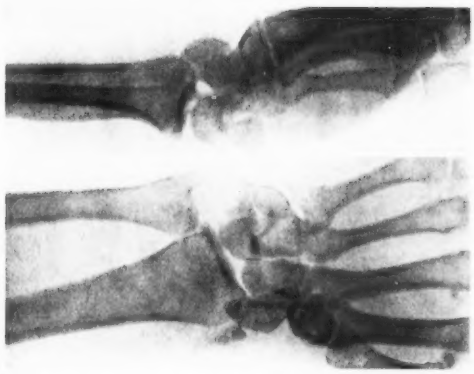

Fig. 2. Complete backward dislocation distal row of carpus. Fracture of scaphoid and radial styloid.

the skin. In 3 of our cases the injuries were due to the fact that the patients fell backward, and landed on their outstretched hands. Two were caused by the kicking back of an automobile crank handle. In all of our cases severe trauma produced the injury.

PATHOLOGY

According to Scudder "osseous union between fragments is the exception and fibrous union the rule." This does not include fracture of the tubercle of the scaphoid. Speed states that following carpal fracture a condition simulating Kuemmel's disease of the vertebra exists; "the traumatized bone dies, is absorbed, a cavity forms and the residue acts as a foreign body in the joint." In our opinion, in addition to the foregoing, much of the disability following carpal fracture is due to damage of the surrounding soft structures. The cartilage, as cartilage elsewhere, has a tendency to heal unevenly and acts as a joint irritant. Neither the synovial membrane nor the tendons crossing the bones escape injury. In addition to the bony damage, we are dealing with a chondritis, arthritis, and synovitis. When the tissues are not placed at rest—so as to allow healing—secondary arthritic changes take place. The result is often a persistently painful wrist with diminished strength and limited motion. Destot truthfully states "a mobile painful wrist is not as good as a good ankylosis." We essentially agree with Scudder and Speed. In our series, however, we find some exceptions. We do have cases which have apparently healed by osseous union, with slight or no breaking down of the cancellous bone structure.

INCIDENCE

According to Speed, fractures and dislocations of the carpal bones occur about 1 in 8 to 10 times as compared to fractures of the wrist region, that is, Collés and kindred fractures.

Hirsch in 1914 stated that there were 1 or 2 scaphoid fractures to every 10 Collés or lower radial fractures (Speed).

Immelmann in 1907 studied 4,000 skiagrams of fractures to find 20 of the scaphoid (Speed).

Todd in 3,000 ambulatory fractures found 9 of the scaphoid (Speed).

Schoch in 150 fractures of the scaphoid, found only 60 of that bone alone (Speed).

Peters saw 32 cases of fractured carpal bones in one year (Speed), as follows:

Fracture of the scaphoid	18
Fracture of the scaphoid and semilunar	1
Fracture of the semilunar	8
Fracture of the semilunar and cuneiform	1
Fracture of the cuneiform	1
Fracture of the os magnum	1
Fracture of the os magnum and cuneiform	1
Fracture of the pisiform	1

Marshall collected 81 cases of carpal bone fractures from the records of the Massachusetts General Hospital. They were:

Simple fracture of the scaphoid	64
Simple fracture of the trapezium	2
Simple fracture of the cuneiform	1
Simple fracture of the semilunar	1
Fracture of the scaphoid plus fracture of the styloid process of the radius	3
Fracture of the scaphoid plus dislocation of the semilunar	5
Fracture of the scaphoid, unciform, styloid process of the ulna, and dislocation of the lunate	1

Fracture of the trapezium plus fracture of the base of the first metacarpal	1
Fracture of the scaphoid plus fracture of the styloid process of the ulna	2
Fracture of the navicular plus fracture of the base of the first metacarpal	1

(Speed)

Bizzaro, in 123 cases of carpal injury, found the scaphoid alone was fractured in 68, and the scaphoid fractured in association with other bone lesions in 38, or a total of 106 scaphoid fractures.

Robinson reported a case of backward dislocation of semilunar.

Carter studied 250 roentgenograms of wrist joint injuries and found 4 per cent were injuries to the carpus, equally divided between scaphoid and semilunar. He had one dislocation of the semilunar associated with fracture of the radius.

Destot in 1921 collected the following cases:

Fracture of the scaphoid	150
Fracture of the semilunar	80
Fracture of the os magnum	4
Fracture of the cuneiform	6
Fracture of the trapezium	4

In our list of cases we found the following:

Fracture of the scaphoid with fracture of radial styloid	1
Fracture of the scaphoid	13
Dislocation of the semilunar	5
Dislocation of the semilunar associated with the fracture of the radius	3
Dislocation of all of the carpals except the semilunar	1
Dislocation of the proximal carpals on the distal	2
Dislocation of the carpus with fracture of the radius	1
	26

The apparent discrepancy is due to the fact that in one of our cases both wrists were involved. It is evident that the scaphoid is the carpal bone most frequently damaged. When the anatomy of the wrist is considered, the reason for the more common scaphoid fracture is obvious. All of our cases were in males; the youngest patient was 17 years old, while the oldest was 53 years of age. In 19 cases the right hand was involved, and the left hand in 17. Again an apparent discrepancy for the reasons above stated.

SIGNS AND SYMPTOMS

Signs and symptoms common to all carpal injuries are swelling, limitation of motion, pain aggravated by motion, and acute tenderness over the site of the lesion.

In fractures of the scaphoid, the acute tenderness is in the tabatière; both active and passive motion is painful with the pain greatly increased when the hand is in dorsal or radial flexion. Indirect pain is elicited by jamming the thumb against the carpus. Radiographic examination of both wrists should always be made, since the existence of a bi-partite scaphoid must be remembered.

In dislocation of the semilunar, disability is almost complete. The wrist and fingers are greatly limited in motion; the fingers are flexed; the dorsal flexion of the wrist is very painful and limited. Extension of the fingers is accompanied by pain. An undue prominence is noted on the anterior surface of the wrist. Radiographic examinations should always be in at least two axes.

Moorhead's axiom, that every crippling or disabling injury at any joint, except the shoulder joint, should be considered a fracture until proved otherwise, is most appropriate at this joint.

TREATMENT OF SCAPHOID FRACTURE

Where there is no displacement of the fragments, the wrist and hand are immobilized with the wrist in dorsal flexion and the fingers flexed. The thumb is also immobilized and held in abduction and complete extension. A cock-up splint of either metal or plaster of Paris extending from just below the elbow to just above the web of the fingers, with an extension for the thumb, is applied. This position is maintained for from 6 weeks to 2 months. Where there is displacement of the fragments, designated by us as Type 1 in contradistinction to the non-displaced or Type 2 cases, steady traction on the fingers with the hand in palmar flexion is employed so as to widen the space between the carpus and the radius. At the same time pressure is made upon the displaced fragment in an effort to force it into its normal position. Failure to secure proper reduction should be followed by immediate removal of the offending bone or the displaced part thereof, else it will act as a foreign body and an irritant to the joint with a resultant limitation of motion and pain. The German surgeons have been inclined to remove the entire scaphoid, while we favor the removal of only the displaced fragment. From the literature one is inclined to believe that the end-results are about the same. However, in cases in which a fracture has existed for some time without being recognized and consequently no attempt at reduction has been made, it is necessary, to obtain good function of the wrist, to remove the entire bone. After reduction is obtained, immobilization for 6 weeks is advisable. After the first 3 or 4 days, massage and baking of the wrist should be carried on daily without removing the splint and without moving the joint. After 6 weeks the splint is removed and a wristlet is worn for a fortnight. With the removal of the splint,

massage, baking, and active motion are employed. The period of disability is from 2 to 4 months.

Codman and Chase believe that non-union is a result of failure to immobilize the wrist properly. However, a certain number of cases—in spite of proper immobilization—do fail to unite and give a weak and painful wrist. This type should be operated upon within 3 months; otherwise, the arthritic changes become quite marked. Decision to operate should not rest with the X-ray findings alone, since X-ray examination may show an apparent non-union and yet the condition may not require operative interference. One should be guided by the physical examination and history.

To expose the scaphoid, we have found the best approach to be by an incision on the dorsum of the wrist over that summit of the radius which exists between the grooves through which the extensor longus pollicis and the extensor indicis tendons pass. With the hand in palmar flexion, the wrist resting upon a sandbag, an incision is made over the point mentioned, to extend 1 inch above and an equal distance below the articular surface of the radius. When the joint is opened, the scaphoid comes into view, since the scaphoid is the first bone to be seen below the articular surface of the radius. This method of approach eliminates the danger of mistaking another of the carpal bones for the scaphoid, a not altogether uncommon occurrence. Through the usual method of approach, that is, through the tabatière, if one is unfortunate enough to tilt the hand radially, the trapezium comes into close contact with the radial styloid and might be unwittingly removed. After removal of the scaphoid or a fragment thereof, active motion is immediately begun.

TREATMENT FOR DISLOCATION OF THE SEMILUNAR

An attempt at closed reduction should always be made. The steps are steady traction on the hand and hyperextension (dorsal flexion) of the wrist. These two steps should be steady and sustained. Then, the wrist is brought into palmar flexion while pressure over the displaced bone is exerted. If the gap between the lower radial articulation and the carpus is sufficiently widened, the offending bone will slip back into place with this manipulation. The wrist is immobilized for 1 week, and then massage, baking, and active motion are instituted.

After two unsuccessful attempts at closed reduction, open operation is indicated. The method of approach is from the anterior surface of the wrist. Incision is made over the summit of swelling, tendons that are encountered are retracted, and the semilunar is exposed.

If the bone can be replaced without much trauma to the joint, it should be done. We have been successful in this closed replacement method in cases seen within 24 hours after the injury.

Where such replacement necessitates greater manipulation and increased traumatism to the joint, then the semilunar is removed. Functional results are good. Early massage and early active motion hasten the restoration of function.

Farr was able to reduce a dislocation of the carpal semilunar, on the day of the accident, with great ease and obtained an excellent result. He states that late reduction or late excision may give good, but not perfect, function. Ten per cent of stiffness and weakness of the wrist usually occurs in late cases.

No attempt at either closed or open reduction of carpal fractures or dislocations should be made without the use of an anæsthetic. While operations under direct infiltration and block anæsthesia have been successfully performed, the writer feels that the best results are obtained with the patient under general anæsthesia.

END-RESULTS

It is the opinion of the writer that 100 per cent perfect end-results following fractures at any joint rarely exist. This is particularly true in carpal fractures or dislocations. In judging our end-results, we follow the method as described by Moorhead:

On the basis of 100 per cent for a perfect result, he allows 60 per cent for function, 20 per cent for contour, and 20 per cent for union. Our study of 25 cases made after a lapse of at least one year from the time of injury, gave the following rating: 49 per cent for function, 12 per cent for union, and 15 per cent for contour, or a total of 76 per cent. It must be remembered that this end-result was not alone for fracture of the scaphoid and dislocations of the semilunar, but also included injuries to the other carpals, some of which were extremely severe and of the crushing type of fracture, some associated with dislocation. Among the fractures of the scaphoid, we had 7 perfect results, while in 5 there was definite interference with joint function. Of these 5, 2 were the result of open operation. Of the 8 dislocations of the semilunar, but 1 gave a perfect result.

Deductions were made for painful joints, limitation of motion, scarring, deformity, and apparent non-union of the fragments. In the painful joints, definite arthritic changes were noted

associated with crepitus on motion of the wrist and fingers.

CONCLUSIONS

1. The scaphoid is more often damaged than all the other carpals.

2. Dislocation of the semilunar is frequently associated with a fracture of either the scaphoid or articular surface of the radius.

3. Osseous union in carpal fractures is the exception; fibrous union the rule.

4. Much of the disability following carpal injuries is due to intra-articular soft structure damage.

5. Early attempts at closed reduction of scaphoid fractures or semilunar dislocations should be made.

6. Failure to obtain proper reduction by closed manipulation should be followed by immediate open operation.

7. Cases of carpal fracture treated unsuccessfully by immobilization should be operated on within 3 months lest irreparable subsequent arthritic changes take place.

8. Carpal fractures, due to the type of bone, do not heal kindly, except fractures of the tubercle of the scaphoid which is definitely osseous.

9. No fracture or dislocation of the carpus ever exists without an associated chondritis, synovitis, and tenosynovitis.

10. Both wrists should always be examined with the X-ray as an aid to diagnosis but the physical findings should be the guide in judging the end-result.

REFERENCES

1. Bizzaro. Surg., Gynec. & Obst., 1922, xxxiv, 574.
2. Codman and Chase. Ann. Surg., 1905, xli, 11.
3. Carter. Ann. Surg., 1925, lxxxi, 532.
4. Destot. Injuries of the Wrist. New York: Paul B. Hoeber, 1925, p. 97.
5. Farr. Ann. Surg., 1926, lxxxiv, 112.
6. Marshall. Boston M. & S. J., 1917, clxxvi, 333–337;
7. Moorhead. Personal communication.
8. Scudder. The Treatment of Fractures. 10th ed. Philadelphia: W. B. Saunders Company, 1926.
9. Speed. Traumatic Injuries of Carpus, p. 65.
10. Spitzka. Gray's Anatomy, 1913, p. 196.

END-RESULTS AFTER BILLROTH I RESECTION OF THE STOMACH AS MODIFIED BY DR. J. SCHOEMAKER

DR. A. A. BUYTENDORP, MARACAIBO, VENEZUELA

TO give a clear idea of the anatomical alterations in stomachs which have been resected according to the method to be discussed, I will give a description of Dr. Schoemaker's technique as described in the *British Journal of Surgery* in 1922.[1]

"The abdomen is opened by a median incision from the sternum to the umbilicus, and the wound is held open by a self-retaining retractor. The duodenum is lifted up and the attachment of the omentum clamped and cut in sections. Two small clamps are placed on the first part of the duodenum, which is then divided by the knife. The distal end is covered with gauze, whilst the proximal end is protected by a little shield, which fits on to the clamp. The remaining portion of the small omentum is clamped and cut in sections, the coronary artery being divided in the last. A large pair of special clamps (Fig. 1) constructed in two portions is then applied to the body of the stomach; the blades of these clamps, which are about five inches long, are curved in about the same shape as the normal lesser curvature of the stomach. When in position the blades extend from a point on the lesser curvature of the stomach opposite to the coronary artery, at the junction of the middle and upper thirds of the stomach, to a point about an inch and a half from the greater curvature of the stomach and two inches from the pylorus. The portion of the stomach between the right end of the stomach clamp and the greater curvature is seized by a pair of small forceps like those for colectomy. The stomach is then cut through by a knife applied close to the clamps, large and small. This frees the pylorus and lesser curvature of the stomach, that are removed. The large stomach clamp consists of two portions. After cutting away the pylorus, the distal portion of the clamp is unscrewed and slipped out, thus leaving a compressed edge of stomach wall rather more than one-eighth of an inch in extent projecting from the remaining portion of the clamp (Fig. 2). This projecting edge is sewn over by a continuous catgut stitch, and the remaining portion of the clamp is then taken away and a second continuous Lembert stitch completes the closure of this portion of the gastric wound. The stomach has now been reduced to a more or less tubular structure, the end of which is closed by one small colon clamp. This is brought into apposition with the duodenum, and after two more clamps have been applied, proximal to the gastric and distal to the duodenal forceps, an end-to-end junction is effected (Fig. 3). The deep surfaces of the viscera are joined by interrupted silk sutures. The terminal clamps are taken off and the whole thickness of the stomach and gut united by a series of interrupted stitches, whilst the anterior layer is completed after the remaining clamps have been removed."

The Billroth I resection is done in all cases of gastric ulcer and also in the cases of duodenal ulcer provided the duodenum can be made movable so that an end-to-end anastomosis can be made. Some surgeons fear that it may not be possible to make the suture at the point where the new lesser curvature and anastomosis meet sufficiently tight to prevent leakage. Dr. Schoemaker has never experienced this difficulty as long as he has been practicing his method. In this method a great part of the lesser curvature together with the ulcer is always removed, so that the so called ulcer-bearing area is excised.

Eighteen patients suffering from ulcer in the stomach and duodenum were examined before and after operation by means of fractional test meals. Several sorts of test meals can be given. In Germany several investigators give an alcohol test meal with the addition of some caffeine and a few drops of methylene blue. As this irritates the stomach too much we prefer the test meal advised by McLean. Finely ground oatmeal is boiled in water and strained through muslin, so that the liquid is clear, is very easily aspirated, and does not tend to block the tube. Instead of the methylene blue, we add tincture of iodine to the different specimens to determine the emptying time of the stomach. As long as there is starch in the specimens, they will show a blue color. In our series we have never had any ill effects from the introduction of the tube. The tube used is the duodenal tube as devised by Rehfuss.

Acid and total acid determinations are made with the ordinary methods, the total chlorides with the Van Slyke method. The number of cubic centimeters of alkali used for the first titration when multiplied by 0.0365 gives the

[1] Brit. J. Surg., 1922, xi, 124.

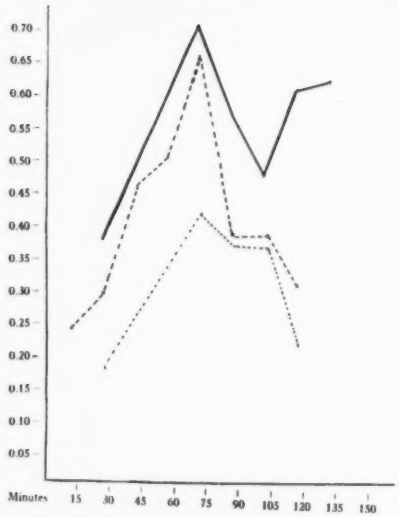

Fig. 1. High acidity in resected stomach.

"free acid" percentage; the number of cubic centimeters used after adding phenolphthalein multiplied by the same factor gives the "combined acid."

The tube is kept in for 150 to 180 minutes. The discomfort of the patients is not great; therefore we cannot speak of a psychic inhibition of the digestive process. The curves thus obtained are, truly speaking, acidity curves and not secretion curves as Katsch and Kalk showed, although some idea of the secretory power of the stomach is obtained.

The normal stomach presents a curve showing a rise in hydrochloric content as the process of digestion begins followed by a fall, as the stomach empties, whereas the total chlorides give a more slowly ascending curve, which very often makes a sharp turn upward at the point at which the acid curves begin to descend. Heidenhain and Pawlow explain this by the fact that neutralization takes place through the alkaline content of the stomach. The most important of these are the mucus of the stomach, the pyloric secretion, which is alkaline, and the regurgitation of the intestinal contents (gall and pancreatic fluid).

The importance of the pyloric secretion is not great; according to Pfaundler it amounts to one-tenth normal sodium hydroxide per 100 cubic centimeters. As to regurgitation of the duodenal contents, a direct relation has not been established between the descending acid curves and the ascending chloride curves. Boldyreff found

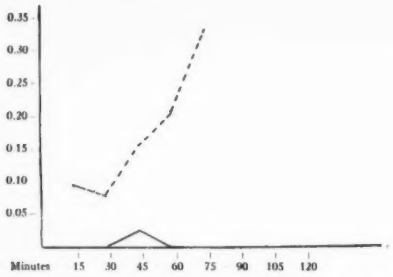

Fig. 2. Patient had a gastro-enterostomy and 8 years later a resection.

that regurgitation of the intestinal fluid takes place under certain circumstances; for instance, in the presence of a great amount of acid or oil in the stomach. Roseman believes that it is more physiological to accept a change in the formation of hydrochloric acid during digestion.

In all the stomachs resected we have always found a subacidity and sometimes a total absence of free chlorides. The total chlorides have been diminished slightly, although we have not always found large amounts of gall or trypsine in the stomach specimens. This contradicts the alkalinizing action of the intestinal contents. Why gall and intestinal contents are found in all specimens will be shown later on.

That even a resected stomach, if too much of the antrum is left, may show high acidity curves is demonstrated in Figure 1. At the second operation in this case no ulcer was found but only a slight stenosis. Stenosis alone, however, does not always cause hyperacidity, as is shown in the curve in Figure 2. This patient first had a gastro-enterostomy; 8 years after that a resection. At the examination of the stomach contents and at X-ray examination after the resection, there was marked retention, although the curve showed strong hypacidity.

The two main factors causing the diminished acid content of the stomach are: the removal of the pyloric mechanism and the removal of the ulcer-bearing area. The pylorus as a regulator of the output disappears. A sphincter may be formed, but an autonomic co-ordinating sphincter is never secured. The manner in which a stomach operated upon by the Billroth I method empties is mainly hydrodynamic in character, especially in the upright position.

As we already have stated, most of the resected stomachs show a slight retention. This might be due shortly after the operation to a postoperative weakening of the muscles, although in some

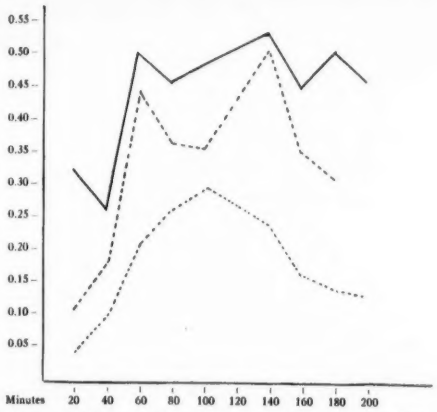

Fig. 3. Differences in acid content before operation.

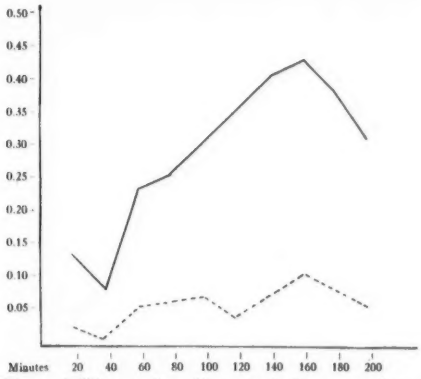

Fig. 4. Difference in acid content after operation.

stomachs observed a long time after the operation a slight retention has been found. Also in X-ray examinations we find retention after the bismuth meal; it is not great, but very often it is evident.

Most authors describe a very rapid emptying time in stomachs resected, and sometimes patients complain of it. This difficulty is very rarely seen in the patients operated upon by Dr. Schoemaker's method. It is also true that complaints of retention are few when the Schoemaker method is used. Excessive retention is caused by two things: first, when the new formed lesser curvature shrinks, the greater curvature becomes relatively too long; and second, when the greater curvature is left too long at operation. As to whether excessive retention may be due to muscular hypotonus I have been unable to decide. One point should be kept in mind and that is that very often in this method of resection we remove the vagus nerve. It courses as a fine white thread through the uppermost part of the lesser curvature. When the lesser curvature is resected high up, the nerve fibers of the remaining part of the stomach are resected. The influence of the vagus on the resected stomach is not yet very clear, but it may be a factor in the production of the relatively longer emptying time in stomachs resected after the technique described. It would be very beneficial to compare the stomachs with and without vagus resections, especially as some surgeons have been obliged to operate again on stomachs which have been resected as the patients complained of the too rapid emptying of the stomach.

Regarding the behavior of the new gastric outlet, in most of the cases reported we find that the new formed pylorus does not work but is always open. Arisz found roentgenologically, by serial exposures of the exit, that it formed a narrow canal, the diameter of which varied between ½ to 3 millimeters. At operation we can easily put at least one finger through the newly formed pyloric canal.

Two things are possible: either a stenosis or the formation of a localized muscular spasm acting in part as a new pylorus. Stenosis may be eliminated as a stenosis would not show changes in width during X-ray exposures. Besides, in one patient suffering from a recurrent ulcer after a Billroth I resection, the radiologist saw an opening the same as that already described, whereas at operation I could very easily pass a finger through the pyloric ring. Arisz described a similar case, in which the patient afterward was operated upon for another reason and in which a finger could very easily be passed through the opening. Forsell and Hellmer attributed this change of tonus to a change in the tonus of the muscularis mucosæ, which because of the nature of the operation is re-enforced at this spot. Goetze describes the mechanism of the new gastric outlet a little differently. He emphasizes the behavior of the duodenal bulb. He found that it filled more readily than in the normal stomach and sometimes became double the normal size. But in most of our cases the bulb is removed.

Very apparently the new outlet does not function as does the pyloric sphincter so that the food flows directly into the duodenum. Although in these stomachs we find that some kind of sphincter is formed, yet there is no comparison between it and the finely regulated mechanism of the normal stomach.

As the food passes more quickly into the duodenum, we find a higher acid content of the duodenal contents, with the result that there is a diminished pepsin action, but as the greatest part of the peptic digestion takes place in the intestine, we must not lay too much stress on this fault in intragastric digestion. Of more importance might be the diminished bactericidal action of the normal acid gastric juice; we haven't seen, however, a great difference in the liability to gastro-enteritis in stomachs resected by the Schoemaker method. In most of the specimens taken from resected stomachs we have found an increased amount of mucus, that is, in stomachs examined a rather short time after operation. In one stomach operated upon 4 years before, I could not detect an increase in mucus. The additional mucus, however, does more good than harm, as it protects the stomach wall against too sharp and rough food. However, in stomachs in which the segment of duodenum left is longer than usual the duodenal bulb is filled by the rapidly inflowing gastric contents. The sphincter of the duodenal bulb although much weaker than the pyloric sphincter presses the food backward into the stomach, thus regulating to some extent the gastric outflow—a mechanism of more importance in the too rapidly emptying stomach.

In stomachs resected after the Schoemaker technique the emptying time is not so rapid as that mentioned by other authors. This has been proved by our fractional test meals and has been described by Arisz roentgenologically. The average emptying time found in our cases is 35 to 45 minutes. Arisz found the time 30 minutes, but when he gave a test meal of barium with potatoes and meat the emptying time became 3 hours, so that we see that the emptying time is dependent upon the sort of food taken.

The secretion time in our series is also much longer than was described by Goetze. Whereas he found an average time of 90 minutes, in our cases it varied between 110 to 160 minutes. As already mentioned, we found in almost all our resected stomachs slight retention; specimens of material retained never showed the presence of free acid, and in 14 of 18 stomachs no acid at all could be found. As to the secretion, we found in all the cases an hypacidity and in one nearly an acidity, whereas the total chlorides did not change much. In one stomach examined 4 years after operation, the same hypacidity existed as at operation. The hypacidity therefore is not a temporary one, as is sometimes the case after gastro-enterostomy. In a series of cases in which gastro-enterostomy had been done Berberich found a subacidity directly after operation, but after 3 years the acid content became normal. This was not mentioned in the series of resected cases. Figures 3 and 4 will give a good idea of the difference in the acid content before and after resection.

SUMMARY

In all cases properly resected, a subacidity was found.

There were no complaints of a too rapid emptying time or of regurgitation.

If the ulcer and the ulcer-bearing area are properly removed recurrence is very seldom found.

A study is still being carried on in this clinic as to the incidence of recurrent ulcers.

FRACTURES OF THE PELVIS[1]

RALPH COLP, B.A., M.D., F.A.C.S., AND ROBERT T. FINDLAY, M.D., NEW YORK

IN this industrial and mechanical age, fractures are not unusual occurrences, and the incidence of certain fractures has increased materially. Fractures of the pelvis are no longer infrequent, and it is the purpose of this clinic to review a series of 35 consecutive cases of pelvic fractures admitted to the Beekman Street Hospital, New York, from January 1, 1925, to October 1, 1928. This group includes not only fractures of the os innominatum but traumatic separations of the symphysis pubis, and the sacro-iliac synchondroses, as these articulations are practically synostoses in the adult.

The ages of the patients varied between 6 and 68 years; the average being about 34 years. As would be expected, over one-half of the patients were between the third and fourth decade, the time of life in which the capacity for physical vigor is high. It is rather interesting to note that there were only three females in this group, but this is easily understandable when the industrial hazard of most feminine occupations is considered.

It is quite natural that the majority of these injuries should occur in those engaged in trades operating at varying heights above the ground. Table I summarizes these occupations, and Table II demonstrates that the more inactive outdoor occupations are associated with less traumatic risk. Table III, however, graphically indicates the traffic hazard in a congested metropolitan area, for although more than one-quarter were engaged in sedentary or indoor occupations, 14 per cent suffered their injuries while going to and from work.

TABLE I.—ACTIVE LABOR, MOSTLY OUTDOORS, WITH CONSIDERABLE RISK OF SEVERE TRAUMA—20 CASES, 57.1 PER CENT

		Cases
1.	Laborers, unqualified	6
2.	Superintendent of laborers	1
3.	Bricklayer	1
4.	Iron worker	1
5.	Truckmen	3
6.	Window cleaners	2
7.	Longshoremen	2
8.	Carpenter	1
9.	Fisherman	1
10.	Seaman	1
11.	Machine hand	1

Over one-half of these patients were injured while working. In other words, over 50 per cent of the cases were subject to the New York State Workmen's Compensation—their employers being responsible and liable for compensation, and many of the remainder came under the heading of "negligence cases." It is quite evident that fracture of the pelvis is of serious economic importance.

The natures of the trauma producing the injury may be of three varieties: injury by falling, crushing, or collision. These three types are enumerated in Tables IV, V, and VI.

The diagnosis of fracture of a pelvis rarely presents difficulty. Fifteen cases were fractures of the ilium, 24 of the pubis. Four cases disclosed fractures of the ischium, and in 1 case the fracture was at the junction of the ischium, pubis, and acetabulum. There were four fractures of the right acetabular cavity and two of the left. There was a separation of the pubic symphysis in four instances. Dislocation, or separation of the sacro-iliac joints, was present in 5 cases.

From the very nature of the mechanism causing the injury, it is quite evident that a fractured pelvis is invariably accompanied by other fractures. This occurred in over 48 per cent of the cases. As a matter of fact, the number of fractures per patient varied from one to eleven. Some of the locations of other fractures were: skull, scapula, humerus, radius, ulna, ribs, lumbar vertebræ, sacrum, tibia, fibula, and astragulus.

Practically all of these patients received other injuries in addition to the fractures, and most of the complications were caused by the initial

TABLE II.—OUTDOOR OCCUPATION, LESS ACTIVE, AND WITH LESS RISK OF TRAUMA—6 CASES, 17.1 PER CENT

		Cases
1.	Schoolboys	3
2.	Policeman	1
3.	Chauffeur	1
4.	Peddler	1

TABLE III.—SEDENTARY OR INDOOR OCCUPATIONS—9 CASES, 25.7 PER CENT

		Cases
1.	Clerks	2
2.	Stenographers	2
3.	Merchants	2
4.	Porter	1
5.	Insurance broker	1
6.	Housewife	1

[1]From the Surgical Service of the Beekman Street Hospital, New York.

TABLE IV.—INJURIES BY FALLING—15 CASES, 42.8 PER CENT

1. Fell 3 feet landing on plank
2. Fell through hatch cover into hold of ship
3. Fell under "L" train
4. Fell 10 feet
5. Fell 12 feet downstairs
6. Fell 1 story down elevator shaft
7. Fell 1 story from scaffolding
8. Fell 35 feet
9. Fell 2 stories from scaffolding
10. Fell from "L" platform to street
11. Fell 60 feet from scaffolding
12. Fell 3 stories
13. Fell 3 stories from window to street
14. Fell 5 stories into skylight
15. Jumped 5 stories down airshaft

TABLE V.—INJURIES BY CRUSHING—13 CASES, 37.1 PER CENT

		Cases
1.	Crushed between two trucks	3
2.	Crushed between truck and platform	2
3.	Crushed between floor and elevator	1
4.	Crushed in elevator	1
5.	Run over by automobile	1
6.	Struck and run over by taxi	1
7.	Run over by horse and cart	1
8.	Run over by truck	2
9.	Taxi overturned, crushing patient	1
10.	Thrown to ground in auto accident	1

TABLE VI.—INJURED BY COLLISION—7 CASES, 20 PER CENT

		Cases
1.	Struck by truck	2
2.	Struck by automobile	1
3.	Struck by street car	1
4.	Street car collision	1
5.	Taxicab collision	1
6.	Struck in groin by boom of fishing boat	1

trauma. The associated injuries included abrasions, contusions, lacerations, and burns of practically all areas of the body; genito-urinary injuries such as rupture of the urinary bladder in 3 cases (one with extravasation of urine), contusion of the bladder in 1 case, temporary paralysis of the bladder in 1, and a rupture of the urethra in 2 cases. Some of the other complications were punctured pleura, traumatic pneumothorax, cerebral concussion, ruptured jejunum, general peritonitis, lumbosacral sprain, compound dislocation of astragulus, hæmatoma of various regions, and abdominal wall infection. These complications added to the fractures already mentioned demonstrates the enormous amount of injury suffered by these patients and shows what an important rôle fracture of the pelvis plays in traumatic surgery.

X-ray examination of these cases, which is always essential, discloses very interesting findings. Displacement and separation of fragments were present in some degree in 28 or 80 per cent of the cases, and absent in 7 or 20 per cent. No displacement was mentioned in 2, slight separation in 6, considerable separation in 2, separation of a chip in 2, complete dislocation of the sacro-iliac joint in 3, subluxation of the sacro-iliac joint in 2, and separation of the symphysis pubis in 5 cases.

The most important symptom of a fractured pelvis is pain. The location of the pain is given in Table VII.

TABLE VII.—LOCATION OF PAIN

			Cases
1.	Generalized pain in pelvis	51.3 per cent	18
2.	Pubic region		1
3.	Iliac region		4
4.	Over symphysis pubis		1
5.	Hip		7
6.	Back		7
7.	Groin		3
8.	Thigh		3
9.	Lower abdomen	17.1 per cent	6
10.	Right calf		1
11.	Foot		1

Tenderness is regularly present. Its location is shown in Table VIII.

TABLE VIII.—TENDERNESS

		Cases
1.	Pelvis in general	19
2.	Abdomen	7
3.	Iliac region	4
4.	Iliac crest region	3
5.	Hip	4
6.	Pubic region	3
7.	At symphysis pubis	3
8.	Back	3
9.	Sacral region	1
10.	Inguinal region	1
11.	Thigh	1
12.	Calf of leg	1
13.	Foot	1
14.	On lateral compression of pelvis	3
15.	Tenderness of pelvis on motion of leg	6

Swelling was visible in a large number of cases. Subcutaneous hæmorrhage was noted as shown in Table IX.

TABLE IX.—SWELLING

		Cases
1.	Hæmatoma of the lumbar region	2
2.	Hæmatoma of iliac region	1
3.	Ecchymosis of buttock	2
4.	Ecchymosis of sacrum	1
5.	Ecchymosis of abdomen	1
6.	Ecchymosis of scrotum	1
7.	Ecchymosis of perineum	1
8.	Ecchymosis of thigh	1
9.	Ecchymosis of hip	3

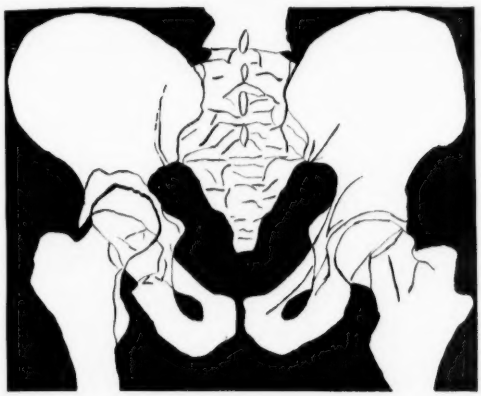

Fig. 1. J. N. K., male, 49 years of age, policeman, thrown in automobile accident. X-ray examination disclosed an 8 months' old fracture of right acetabulum and depression of acetabulum so that the head was driven one-half way into pelvis. Now healed. He also received a laceration of the sciatic nerve and developed a traumatic sciatic neuritis. The result in this case was poor. Although there was 1½ inches shortening, the movement of the hip joint was perfect. His poor result was due to nerve injury with atrophy of muscles, weakness, pain, etc.

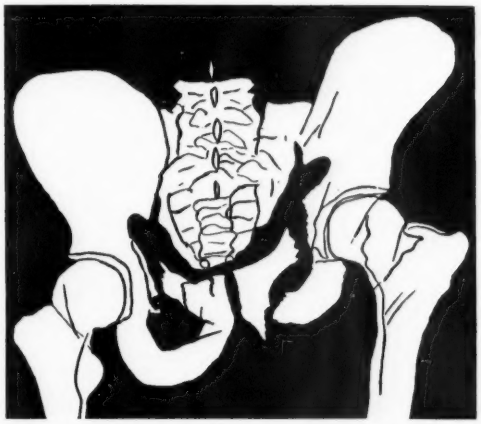

Fig. 2. L. S., male, 32 years of age. X-ray examination disclosed 6 fractures of the pelvis as follows: (1) at junction of horizontal ramus of left pubis and ischium with ¾ inch separation; (2) at descending ramus of left pubis with ¾ inch separation; (3 and 4) fractures of the horizontal ramus of the right pubis; (5) fracture of descending ramus of right pubis; and (6) fracture at juncture of right ischium, pubis, and obturator foramen with ½ inch separation. Upward and backward displacement of left pelvis. This man fell from the fifth story into a skylight while cleaning windows. Beside the multiple fractures of the pelvis he sustained a fractured sacrum and 2 transverse vertebral processes. He developed a hæmolytic streptococcus infection of a hæmatoma of the sacral region which was drained several times. He died after 41 days of multiple fractures plus infection plus hæmorrhage from the abscess cavity.

Shock was severe in 18 cases. Two cases were unconscious at the time of admission, the unconsciousness, however, being caused by the associated injuries.

Six of the patients, or 17 per cent, were unable to void, 2 unable to use their legs, and 1 had a temporary anæsthesia of the feet and legs.

The majority, upon arrival at the hospital, were completely disabled; 11 had practically complete disability and 22 were partially disabled.

Crepitus is rather an unusual physical sign in this type of fracture and was present in only 3 cases.

The right foot was externally rotated in a case of the fracture of the left ischium with a spreading of the left sacro-iliac joint.

Voluntary limitation of motion of the extremities was mentioned in 4 cases. It was rather interesting to note that in many of these cases certain abdominal symptoms were present which were probably due to reflex spasm incident to fractures of the pelvis. Abdominal examination revealed muscle spasm in 8 cases, distention in 3, tenderness in 7, and ecchymosis of the lower abdominal wall in 1.

Bladder injury has always been thought to be quite common in this type of fracture. Hæmaturia was seen in 3 cases, in one of which almost pure blood was removed by catheter. Two cases

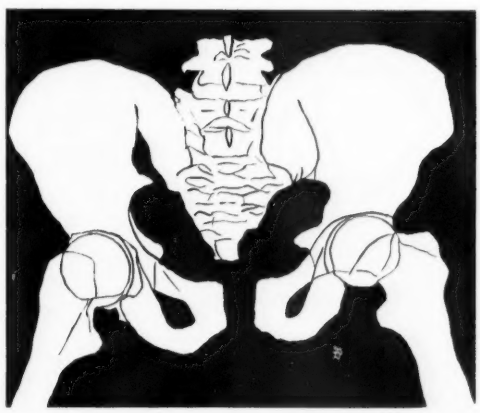

Fig. 3. B. D., male, 45 years of age. X-ray examination disclosed a separation of right sacro-iliac joint and symphysis pubis. This laborer fell 35 feet and also received fractures of 2 transverse vertebral processes as well as a ruptured bladder and many other injuries. He died a cardiac death after 29 days in the hospital.

Fig. 4. R. R., male, 32 years of age. X-ray examination disclosed an oblique fracture through right ilium from sacro-iliac joint to crest. Comminuted fracture of crest. Fracture of both rami of pubis. Practically no displacement. This man was crushed between two trucks. His recovery was uneventful and he left the hospital after 54 days. Two months later he complained of pain in the pelvis and inability to do hard work as yet. Nothing further has been heard from him.

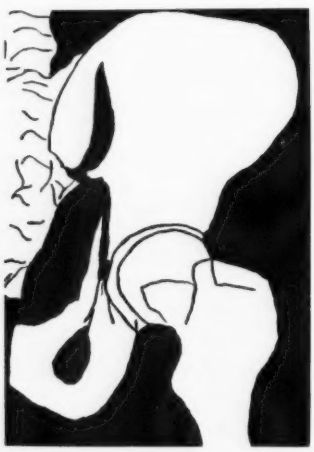

Fig. 5. V. B., male, 42 years of age. X-ray examination disclosed a fracture of left ischium, spreading of left sacro-iliac joint. This man, an ironworker, fell two stories from a scaffolding and received in addition to the above a fracture of the base of the skull. After 16 hours he died from the fractured skull plus shock.

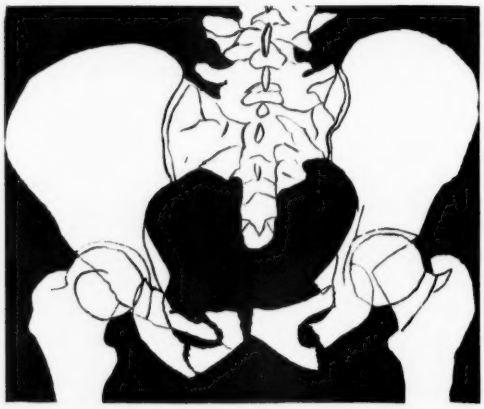

Fig. 6. M. D., female, 19 years of age. X-ray examination disclosed a fracture of both rami of left pubis. Fracture through ramus of right pubis near ischium and through ascending ramus at body and acetabulum. Also comminution of pubis. Right side, good position. Left pubic fragments considerably separated. Left ilium displaced upward. This young lady was in a taxi cab which overturned, giving her the above injuries plus fractures of the sacrum and of an articular vertebral process. She was walking in 7½ weeks and left the hospital after 58 days. She was seen and examined a few weeks ago, practically 4 years since injury, and found to be in excellent health with no symptoms or signs of any injury.

were accompanied by extravasation of urine, one to the scrotum, and one to the inguinal region. Injection tests were done for evidence of bladder injury in 4 cases. In 1, air was injected, and in the roentgenograms air could be seen in the bladder and also in the perineum outside of the bladder. The installation of sterile saline in 3 other cases showed that the bladder was intact.

The treatment of these cases resolves itself in the treatment of the fracture and the complications. Rest in bed is the most important part of the treatment for the fracture and in the majority it was practically the only treatment administered. All patients were put flat on their backs in bed and kept there on an average of 30 days. Patients were allowed up gradually, first being rolled, and later turned in bed, then permitted to use a wheel chair, and finally they were allowed to walk. The length of time after injury that patients were allowed to walk averaged 38 days. Callus was demonstrated by the X-ray in some cases before the patient was allowed to walk. The patients in whom displacement and comminution of the fragments were marked were kept in bed for the longest period of time. Two patients were kept on fracture boards and one on an air mattress. The pelvis was strapped in 2

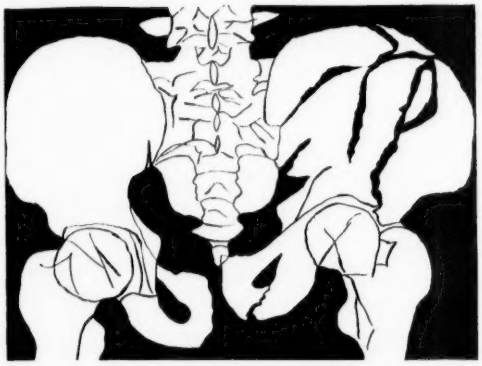

Fig. 7. S. B., male, 38 years of age. X-ray examination disclosed 3 fractures of left ilium, subluxation of sacro-iliac joint. Separation of symphysis pubis. Fracture of left acetabulum. Left ilium rocked backward. This man fell from an elevated railway to the street receiving, besides his fractures of the pelvis, fractures of 2 ribs and a traumatic pneumothorax. He was kept in the hospital 57 days, 47 of which were in bed. Over a year later he was at sea, reported to be in good physical condition.

instances, and 2 patients were fitted with supporting elastic belts. A Balkan frame with traction on the leg to pull down a posterior displacement of the ilium was used unsuccessfully in 1 case.

This study certainly emphasizes the simplicity of treatment of fractures of the pelvis. It should be particularly noted that no patient was operated upon, that no casts were used, and that traction was employed in only one instance. Even the advisability of an air mattress and fracture board is an open question. The indication for their use is purely dependent upon the patient's comfort.

During the past year we have been inclined to use elastic pelvic supports, and we believe that the support is highly advisable, especially in complete, comminuted, and separated fractures. We feel that a polo belt may be used to great advantage.

Operations were performed only when complicating injuries to soft parts required it. Brief outlines of these case histories follows:

CASE 1. H. H., male, aged 43 years, was injured in a street car collision. He received a fracture at the junction of the left ischium, pubis, and acetabulum, with separation of the left sacro-iliac joint; also rupture of the urinary bladder and prostatic urethra with extravasation of urine and a hæmatoma of left thigh. He was operated upon after reacting somewhat from shock. A tear 1½ inches long was found in the anterior wall of the bladder extraperitoneally, with extravasation of blood and urine into the rectus muscles. The bladder and the space of

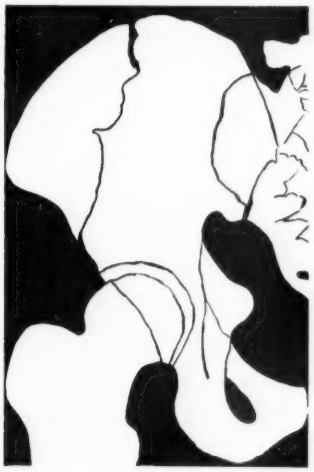

Fig. 8. M. H. H., male, 32 years of age. X-ray examination disclosed a long fracture of right ilium with ¼ inch separation. This man was run over by a truck while crossing the street and received, in addition to the above, fractures of 4 transverse vertebral processes. With only 3 weeks in bed, the patient left the hospital in a little over a month. We were never able to trace him.

Fig. 9. P. D., male, 35 years of age. X-ray examination disclosed a transverse fracture of body of left ilium, no separation. This patient was crushed in an elevator and besides the above received fractures of ribs, the scapula, and of a transverse vertebral process. Notwithstanding having had cerebral concussion and lobar pneumonia, he was getting along nicely and was still in the hospital at the time of writing, 1½ months after injury.

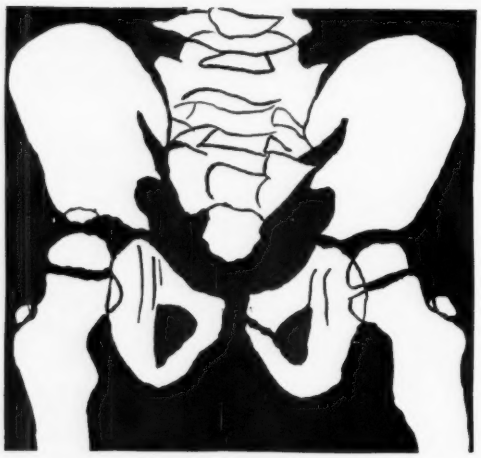

Fig. 10. J. C., male, 6 years of age. X-ray examination disclosed a fracture of right obturator foramen, no displacement. This schoolboy was run over by a horse and wagon. He also sustained a fractured humerus and other injuries. His parents removed him from the hospital after 32 days of satisfactory progress and nothing has been heard from him since.

Retzius were drained. The abdominal wound became infected and was opened for drainage. A secondary operation was done later. As the infection was not controlled, a peritoneal urethotomy, retrograde catheterization of the urethra, and perineal drainage of the space of Retzius were performed. A blood transfusion was given on the seventh day, but the patient died on the fourteenth day from infection.

Case 2. B. D., male, aged 45 years, fell 35 feet while working. He sustained a separation of right sacro-iliac joint and symphysis pubis, fractures of both transverse processes of the fourth lumbar vertebra, a rupture of the bladder, and an enormous hæmatoma of the sacral region. A suprapubic cystotomy was done on the second day, two rents in the anterior bladder wall were found, both extraperitoneal, one measuring three-quarters of an inch, the other one-half inch. One tear was sutured, the other used for drainage. The hæmatoma of the back was incised and drained. The bladder and surrounding tissues became infected and additional stab wounds in the groin were necessary for adequate drainage. The patient did well until cardiac decompensation occurred. This would not yield to treatment and death occurred on the twenty-ninth day after the injury.

Case 3. R. D., male, aged 18 years, fell under an "L" train, receiving multiple fractures of the left ilium and a severe crushing injury of the abdomen. His condition was extremely poor. No definite abdominal diagnosis was arrived at until the next day, when at operation two perforations in the jejunum with acute diffuse peritonitis were found. The patient died 3 hours after operation.

Case 4. F. S., male, aged 50 years, was crushed between two trucks, sustaining a fracture of the right pubis with deformity, a fracture of the right tibia and fibula, rupture of urethra, and contusions and abrasions of hips. He was operated upon with a provisional diagnosis of ruptured bladder. None was found, so a suprapubic cystotomy was

TABLE X.—OCCUPATION SINCE INJURY

		Cases
1.	Same type of work as before injury	6
2.	Less strenuous work	3
3.	Not able to work since accident	2
4.	Not working because still receiving compensation	2
5.	Not working because of inability to find work	1
6.	At sea, apparently working	2
7.	Thought to be doing hard work	1
8.	Working until injured again, now in another hospital for some other injury	1
9.	Unknown	1

TABLE XI.—LATE SYMPTOMS

1. Pain in right iliac region on sneezing or coughing, or prolonged weight bearing.
2. Slight muscular weakness of left leg.
3. Slight pain in pelvis in bad weather.
4. Slight continuous pain in back.
5. Slight pain in right hip and at region of right ischial tuberosity after resting, improved on motion or walking.
6. Slight pain in back after long walking or running.
7. Slight pain in right hip, worse with rest, improved on walking.
8. Slight pain in region of sacro-iliac joints.
9. Increased pain on stooping, improved on walking.
10. Pain in right inguinal and lumbosacral regions, especially on resting, improved on walking.

performed. A secondary operation was done 4 days later by perineal section—anastomosis of the urethra. The patient voided *per urethra* on the fifty-third day and had an excellent result.

When the patients were discharged from the hospital, 21 were in excellent condition, 1 was fairly good, 1 was improved, 2 were fair, and in 3 the result could not be accurately determined. One case had a poor result.

Six of the 35 patients died in the hospital, making a mortality of 17 per cent. The causes of death may be enumerated as follows:

First case: (a) multiple fractures of the pelvis, (b) hæmatoma of the back which was incised and drained, (c) streptococcus hæmolyticus infection, and (d) secondary hæmorrhage from back incision. Length of life, 41 days.

Second case: (a) multiple fractures of the pelvis, (b) rupture of the urinary bladder, (c) rupture of prostatic urethra, and (d) infection of abdominal and perineal incisions and of the bladder. Length of life, 14 days.

Third case: (a) fracture of the left ilium, (b) ruptured jejunum, (c) acute diffuse peritonitis, and (d) shock. Length of life, 27½ hours.

Fourth case: (a) separation of right sacro-iliac joint and symphysis pubis, (b) fracture both transverse processes of fourth lumbar vertebra, (c) rupture of urinary bladder, (d) hæmatoma of sacral region, (e) abdominal wound infection, and (f) cardiac decompensation, which was the ultimate cause of death. Length of life, 29 days.

Fifth case: (a) multiple fractures of the pelvis and (b) fracture of the base of the skull. Length of life, 16 hours.

Sixth case: (a) multiple fractures of the pelvis, (b) fracture of transverse process of fifth lumbar vertebra, (c) fracture of right sacrum, (d) compound fracture-dislocation of right astragulus, (e) dislocation of right ankle joint,

(f) ruptured urinary bladder, and (g) shock. Length of life, 9 hours.

Thus it will be seen that all of the fatal cases were definitely complicated, and that the fractures of the pelvis played but small part in the causation of death. The length of time in the hospital before death varied between 9 hours and 41 days; the average being 14.36 days.

Eighteen, or 75 per cent, of these cases were followed after discharge. We were unable to follow 6 cases. Eleven were in good condition, 2 in fair, 1 in poor, and 4 undetermined. Table X shows the type of work they are doing.

The late symptoms which these patients complained of are enumerated in Table XI. Pain seems to be the chief complaint.

Physical examination disclosed only slight deformity and bulging of the pelvis on one side, slight tenderness at the site of the fracture, and in one case there was shortening, this occurring in a fracture of the acetabulum. So, excellent results were seen in 6, good results in 9, in 2 of these there was a poor result due to a complication of the fracture, in 1 case the result was poor, and in 2 insufficient time has elapsed to determine the end-results.

THE VALUE OF THE WALCHER POSITION IN CONTRACTED PELVIS

With Special Reference to its Effect on the True Conjugate Diameter

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THE Walcher position, or the dorsal posture with the hips at the edge of the table and the lower extremities hanging over, was used empirically to facilitate labor by Mercurio in 1589 and Melli in 1738.

In his first communication in 1889 on the effect of posture on the measurements of the pelvis, Walcher (30) reported measurements made on six pregnant women. They showed the diagonal conjugate to be increased from 8 to 13 millimeters by placing the woman on the edge of the table with the legs hanging down as compared with the measurements made with the legs flexed against the body. In his subsequent articles (31), he re-stated and commented on these findings without presenting new data. He believed that the softening of the pelvic joints during pregnancy permits the sacrum to become movable and allows the innominate bones to rotate downward so as to enlarge the diameters of the inlet.

In an article published recently (17), I offered the following conclusion among others:

"The Walcher position increases the anteroposterior diameter of the inlet from 0.5 to 1.0 centimeter. When the pelvis is slightly contracted or the fetal head is a little oversized in a normal pelvis, the use of this position often facilitates engagement."

To my surprise, I received a number of letters from physicians denying any material lengthening effect of the Walcher position upon the true conjugate diameter and, in some instances, any benefit in facilitating engagement of the presenting part. For this reason, I thought it advisable to review the literature on the effects of the Walcher position, notwithstanding the fact that practically all authors of textbooks on obstetrics concede its value in cases of relatively contracted pelvis.

THE ACCEPTED VIEW

Polak states the generally accepted opinion as follows: "The posture of the patient has some influence on the diameters of the bony pelvis. When the thighs are extended, as in the Walcher position, the upper end of the sacrum moves backward, while the symphysis is lowered and the brim is slightly increased in its anteroposterior diameter."

According to Shears, "the engagement of the head in the pelvic brim is sometimes favored by placing the patient in the Walcher position. . . . The space is small, 1 to 8 millimeters, according to Pinzani, and few patients can be induced to maintain it for a long time, but in doubtful cases it is worth trying."

Edgar offers the following explanation: "The mechanism of the Walcher position is dependent upon the motion of the sacro-iliac synchondrosis, and is explained as follows: The weight of the limbs hanging from the edge of the table causes the ilia to rotate forward and downward around the transverse axis of the joint. Thus the angle made by the plane of the brim with the horizon is increased, and consequently the symphysis pubis is brought a little forward and downward, and a little further from the sacrum."

Tweedy and Wrench describe the effects of the Walcher position as follows: "The weight of the lower limbs rotates the pelvis on the sacro-iliac joints so that the pubic bone sinks. In this way the true conjugate is actually lengthened by half an inch, and this addition is of the greatest value."

Williams states that in cases of contracted pelvis in which engagement fails to occur after complete dilatation of the cervix the patient should be placed in Walcher's position "for as long a time as she will bear it." In many cases this "will bring about a lengthening of the anteroposterior diameter of the superior strait sufficient to permit engagement."

DeLee discusses the Walcher position as follows: "The true conjugate may be lengthened from 6 to 12 millimeters, especially in flat pelves. . . . In a few cases I have gotten the head to enter the pelvis by this procedure, but it is very painful, and few women can be compelled to keep it up over 20 to 40 minutes. My results, curiously, have been better by the use of the exaggerated lithotomy position, which is theoretically contraindicated because it contracts the inlet."

REVIEW OF LITERATURE

Klein, in 1891, substantiated Walcher's finding that the anteroposterior diameter of the pelvis can be altered by change in position, and that it is longest in the position described by Walcher.

By measurements on the cadaver, however, he found that the conjugata vera was not increased in the Walcher position more than 0.64 centimeter in any type of pelvis. This increase was found in flat pelves. In normal pelves it was increased 0.54 centimeter. The average increase in 29 cases, including normal and abnormal pelves, was 0.58 centimeter. Walcher pointed out, however, that Klein's measurements were made on the cadavers of non-pregnant women, instead of on pregnant women, and that this fact might account for the lesser change in the true conjugate.

Duehrssen, in 1893, stated that his measurement of the conjugata vera in 6 cases in the dorsal and in the Walcher positions showed that in the latter the diameter was increased in length from 0.5 to 1.2 centimeters; in all but 2 cases, 1 centimeter or over. The greatest difference was found in a case with rachitic flat pelvis. He believed that this increase in the conjugata would be of definite advantage in cases of labor with contracted pelvis in which the head cannot be brought down into the pelvis rapidly enough in the usual dorsal position.

Kalt, in 1894, reported 3 cases of narrow pelvis (conjugata vera 8 to 9 centimeters) in which the head could not be brought down into the pelvis until the patient was placed in the Walcher position. By this method a normal, living child was delivered in each instance.

Fehling, in 1894, reported 3 cases of contracted pelvis in which the head was brought down into the pelvis and a living child was delivered by means of the Walcher position. The conjugata vera in these cases was not increased more than 6 to 8 millimeters by the Walcher position in comparison with the measurement in the dorsal position.

Wehle, in 1894, reported 25 cases of contracted pelvis in which version and extraction were used in combination with the Walcher position. Of the 25 babies delivered, 3 were born dead and 3 died within a few days after birth; but, in one of the latter cases, death could not be attributed to the method of delivery. Thus, of 25 children, 20 (80 per cent) were safely delivered, as compared with 68.4 per cent delivered previously by version and extraction without the use of the Walcher position. The transverse diameter of the head was in all cases larger than the conjugata vera measured in the dorsal position.

Fothergill, in 1896, reported 11 cases of contracted pelvis in which the Walcher position resulted in the birth of a living child. In measurements of about 30 pregnant women made by two independent investigators with the women in the lithotomy and the Walcher positions, it was found that the average increase in the diagonal conjugate in the latter position was 1 centimeter. In 6 women with small pelves, the average increase in the Walcher position was 0.93 centimeter.

Huppert, in 1898, reported 28 cases of contracted pelvis in parturient women in which the Walcher position was used as an aid to delivery. In 18 of these cases a living child was delivered without operative interference. The conjugata vera in these cases measured in the dorsal position varied from 6.5 to 9 centimeters. The best results were obtained with flat pelves. A smaller number of patients with generally contracted pelves were delivered by this method. In every case the head of the child was larger by 0.5 to 1.5 centimeters than the conjugata vera measured in the dorsal position.

Kuettner, in 1898, described a method for making plaster casts of the pelvis from the cadaver in various positions. From a study of the measurements of the pelvis by this method, he found that the pelvis of the pregnant or puerperal woman showed definite changes in form and measurement in different positions. It was observed that the anteroposterior diameter of the pelvic inlet could be increased from 0.9 to 1.4 centimeters by the Walcher position; while, by flexion instead of extension of the thighs, the diameter of the outlet was increased. The transverse diameter was not altered in either case.

Cutts, in 1899, maintained that, if there is to be any gain in the measurement of the superior strait, it must be sought in the principle set forth in the Walcher position: i.e., the extension of the ilium on the sacrum. He noted that examination of the skeleton shows that the promontory of the sacrum is superior and anterior to the line representing the axis of rotation of the sacro-iliac joints. When the thighs are extended, as in the Walcher position, the iliopubic arch is rotated on the sacrum, so as to bring the symphysis pubis farther from the sacral promontory. "In other words marked extension causes a lengthening of the true conjugate and, therefore, aids engagement of the head in the superior strait, while marked flexion causes a lengthening of the anteroposterior diameter of the inferior strait."

Examining a female cadaver at the University of Minnesota laboratory, Cutts found that by actual measurement the true conjugate was 4.25 inches with the subject lying flat on the back.

In the Walcher position it was increased to 4⅜ inches. With marked flexion, it was diminished to 4 inches, making the difference in the measurement between complete flexion and extension ⅜ inch. Cutts believed it quite probable that this measurement would be farther increased in the living pregnant state. While the difference of an eighth of an inch in the true conjugate between the dorsal position and the extended Walcher position is not much, yet it may be enough to aid materially in a high forceps operation. Cutts concluded that "at least some cases of dystocia at the superior strait will be benefited by the Walcher position."

Dickinson, in 1899, from his study of the effect of posture on the shape of the pelvis in late pregnancy, came to the conclusion that the hanging dorsal posture should be employed to obtain the longest conjugate at the inlet and that the gain is nearly 1 centimeter.

Dice, in 1911, discussing methods of determining the relative size of the head and the pelvis prior to term, stated: "If the patient is put in the Walcher position, the maximum enlargement of the inlet is secured."

Christiani, in 1913, reported that in a series of 45 cases of contracted pelves the Walcher position was of definite aid in bringing the fetal head down into the pelvis in 11 instances. In 9 cases it had no effect on the progress of labor. He believed that it should be tried in any case in which the discrepancy between the size of the pelvic inlet and the fetal head is not too great and progress of the head is stopped at the inlet. In some cases, he thought that the position not only facilitates the passage of the head into the pelvis but also appears to strengthen the pains.

Martin, in 1925, stated that the Walcher position aids delivery in cases of contracted pelvis. He considered that enlargement of the pelvic diameter by only a few millimeters may be sufficient to allow the head to pass, especially with the aid of proper manipulation.

Krukenberg, in 1926, noted that the Walcher position is of definite value as an aid to labor when there is but slight disproportion between the size of the head and of the pelvis. In such cases, the increase of 0.75 to 1 centimeter thus obtained in the conjugata vera is sufficient to aid delivery. At any rate, he believed that the effect of this position should be tried, as it does no harm.

DIRECT MEASUREMENT OF THE TRUE CONJUGATE

One source of contradiction with regard to the effects of the Walcher position on the conjugata vera arises from the methods clinically employed to determine this diameter. It has been generally assumed that an accurate estimation of the true conjugate can be made by measuring the diagonal conjugate and by making an appropriate deduction according to the estimated height, thickness, and inclination of the symphysis pubis. Yet some recent observations would suggest that this rough method of judging the true conjugate may sometimes be inaccurate.

Dr. J. Bay Jacobs, of Washington, has devised a new obstetric inclinometer, by which the obstetrical diameters, the inclination of the pelvis as a whole, and the angulation of the various planes may be read directly on the instrument and with great precision. With this inclinometer he visualizes the interior of the pelvis in terms of an *obstetric triangle*, formed by the symphysis pubis, the true and the diagonal conjugate.

"Studies with the inclinometer," writes Jacobs (15), "indicate that our ordinary method of computing the true from the diagonal conjugate has frequently caused an underestimate of the degree of pelvic contraction. In some instances, what appears to be a border-line case proves to be one of absolutely contracted pelvis when studied with the aid of the obstetrical inclinometer."

At my request, Dr. Jacobs (16) was kind enough to make observations with his inclinometer in 10 cases of contracted pelvis. I regard his findings as of great importance, since they give direct measurements of the true conjugate; whereas, in most previous reports, the true conjugate was estimated from the diagonal by the ordinary clinical method. I quote from Dr. Jacobs' letter:

> Of a series of 10 patients whose measurements were taken with the obstetrical inclinometer, 9 had moderate and 1 absolute pelvic contraction. The observations were made with the patient first in the dorsal recumbent and then in the Walcher position.
>
> In all cases the Walcher position caused an increase in the diagonal conjugate, varying from 0.5 to 2 centimeters. In the majority, however, the increase was 0.5 to 1 centimeter.
>
> The effect of the Walcher position on the true conjugate, however, depends on the consequent variation in size of what I term the obstetric angle (included between the symphysis pubis and the diagonal conjugate) and the lengthened diagonal conjugate.
>
> The change in the length of the true conjugate varied from minus 0.3 centimeter to plus 2.1 centimeters. In one case, the Walcher position caused a decrease of 0.3 centimeter in the conjugata vera; in another case, there was no change. In the 8 remaining cases the true conjugate was increased by 0.3, 0.7, 0.95, 1.1, 1.6, 1.8, 1.9, and 2.1 centimeters, respectively.

Dr. Jacobs' observations are important, because they show by exact measurement of the

true conjugate that the anteroposterior diameter of the inlet is generally increased by the Walcher position but that it may sometimes be unaffected or even slightly reduced.

ROENTGEN PELVIMETRY

Another method of making exact measurements of the inlet that might be utilized for studying the effects of the Walcher position, and probably will be in the near future, is roentgen pelvimetry. Satisfactory methods proved by comparison with the cadaver to be exact have been described by Bell; Thoms; Dorland; Heublein, Roberts, and Ogden; and by Coats.

Concerning his observations with roentgen pelvimetry, Thoms (28) writes as follows:

> When one considers the quite inadequate information concerning the superior strait that is derived from the ordinary methods of pelvimetry, it is surprising that more attention has not been given to pelvimetry by means of the roentgen ray. I think it is safe to say that the maneuver of most value today is the one that was described nearly 200 years ago by William Smellie. I refer to the method of estimating the diagonal conjugate diameter. The other maneuvers, usually practiced in estimating the size of the pelvic inlet, are inexact and unreliable. Not only does roentgen pelvimetry afford an exact knowledge of the anteroposterior diameter, but the transverse diameter also becomes available.

MANNER OF USING WALCHER POSITION

To be effective, the Walcher position must be maintained sufficiently long. If the patient's legs are allowed to hang over the table without support, this posture is likely to prove uncomfortable and cannot be maintained for more than 5 to 10 minutes. However, if the feet are supported properly, it may be held without too great discomfort for as long as 45 minutes.

In order that the patient may be kept in the Walcher position long enough to facilitate engagement of the presenting part, I have designed and utilized an obstetric table equipped with a shelf on a sliding rack that can be adjusted exactly to fit the height of the patient (17). In the home, the patient can maintain the Walcher position comfortably by placing her feet on a foot-stool or cushion adjusted to the proper height.

SUMMARY

The consensus of opinion as stated in authoritative textbooks on obstetrics is that the use of the Walcher position in contracted pelvic conditions lengthens the anteroposterior diameter of the inlet sufficiently to help engagement. From a careful study of the literature and observations on 7 cases by direct measurements of the true conjugate with an obstetrical inclinometer, it would appear that this view is substantially correct.

However, several qualifications should be stated. In a minority of cases, the Walcher position may not change the length of the conjugata vera or even reduce it. Also, it is necessary to bear in mind that estimations of the true conjugate from the diagonal may be misleading and what appears to be a case of relatively contracted pelvis may really be one of absolute contraction.

Nevertheless there is no valid reason to doubt the efficacy of the Walcher position in many cases of minor degrees of pelvic contraction. Its use frequently avoids the necessity of surgical intervention.

BIBLIOGRAPHY

1. Bell, J. W. Pelvioradiography after Fabre's method. Am. J. Obst. & Gynec., 1921, ii, 616.
2. Christiani, J. Die Walchersche Haengelage. Wien. klin. Rundschau., 1913, xxvii, 449, 465, 485.
3. Coats, A. J. Value of the roentgenogram in pelvimetry. Northwest. Med., 1927, xxvi, 542.
4. Cutts, R. E. A discussion of the Walcher position. Ann. Gynæc. & Pediat., 1899–1900, xiii, 594.
5. DeLee, J. B. The Principles and Practice of Obstetrics. 5th ed. Philadelphia: W. B. Saunders Company, 1928, p. 764.
6. Dice, W. Choice of delivery in moderately contracted pelvis. Am. J. Obst., 1911, lxiii, 459.
7. Dickinson, R. L. The Walcher, the Trendelenburg, and the Mercurio postures in midwifery. Am. J. Obst., 1899, xxxix, 751.
8. Dorland, W. A. N., and Hubeny, M. J. The X-ray in Embryology and Obstetrics. St. Paul: Bruce Publishing Company, 1926.
9. Duehrssen. Diskussion ueber Symphyseotomie. Verhandl. d. deutsch. Gesellsch. f. Gynaek., 1893, v, 46.
10. Edgar, J. C. Edgar's Practice of Obstetrics. 6th ed. Philadelphia: P. Blakiston's Son and Company, 1926, p. 617.
11. Fehling, H. Ueber Verwendung der Haengelage nach Walcher zum Eintritt des vorangehenden Kopfes. Muenchen. med. Wchnschr., 1894, xli, 861.
12. Fothergill, W. E. Walcher's position in obstetrics. Brit. M. J., 1896, ii, 1290.
13. Heublein, A. C., Roberts, D. J., and Ogden, R. T. Roentgen pelvimetry after the Thoms method with a simplification of technique. Am. J. Roentgenol., 1928, xx, 64.
14. Huppert, L. Die Bedeutung der Walcher'schen Haengelage. Arch. f. Gynaek., 1898, lvi, 179.
15. Jacobs, J. B. The obstetric inclinometer. Am. J. Obst. & Gynec., 1928, xv, 689.
16. Idem. Personal communication.
17. Jarcho, J. The rôle of posture in obstetrics. Surg., Gynec. & Obst., 1929, xlviii, 257.
18. Kalt, A. Die Walcherische Haengelage. Cor.-Bl. f. schweiz. Aerzte., 1894, xxiv, 652.
19. Klein, G. Zur Mechanik des Ileosacralgegends. Ztschr. f. Geburtsh. u. Gynaek., 1891, xxi, 74.

20. KRUKENBERG, H. Anatomie und Statik des Beckens; Biologie und Pathologie des Weibes by Halban and Seitz, vol. 7, pt. 2. Berlin and Vienna: Urban & Schwarzenburg, 1926.
21. KUETTNER, O. VON. Experimentell-anatomische Untersuchungen ueber die Veraenderlichkeit des Beckenraumes Gebaerender. Beitr. z. Geburtsh. u. Gynaek., 1898, i, 210.
22. MARTIN, E. Das enge Becken. Beihefte z. Med. Klin., 1925, xxi, pt. 2.
23. MELLI. Cited by DeLee (5).
24. MERCURIO. Cited by DeLee (5).
25. POLAK, J. O. Manual of Obstetrics. 2d ed. New York: Physicians and Surgeons Book Company, 1922, p. 113.
26. SHEARS, G. P. Obstetrics. 2d ed. Philadelphia: J. B. Lippincott Company, 1917, p. 535.
27. THOMS, H. Outlining the superior strait of the pelvis by means of the X-ray. Am. J. Obst. & Gynec., 1922, iv, 257; Pelvimetry of the superior strait by means of the roentgen ray. J. Am. M. Ass., 1925, lxxxv, 253; X-ray pelvimetry; a simplified technique. Surg., Gynec. & Obst., 1927, xlv, 827; A new method of roentgen pelvimetry. J. Am. M. Ass., 1929, xcii, 1515.
28. THOMS, H. A new method of roentgen pelvimetry. J. Am. M. Ass., 1929, xcii, 1516.
29. TWEEDY, E. G., and WRENCH, G. T. Practical Obstetrics. 5th ed. New York and London: Oxford University Press, 1925, p. 374.
30. WALCHER, G. Die Conjugata eines engen Beckens ist keine konstante Græsse. Zentralbl. f. Gynaek., 1889, xiii, 892.
31. Ibid. Die Veraenderlichkeit der Conjugata. Verhandl. d. deutsch. Gesellsch. f. Gynaek. 1892, iv, 446; The influence of the position of the woman on the form and dimensions of the pelvis. St. Louis Cour. Med., 1899, xxi, 341.
32. WEHLE, J. Die Walcher'sche Haengelage. Arch. f. Gynaek., 1894, xlv, 323.
33. WILLIAMS, J. W. Obstetrics. 5th ed. New York and London: D. Appleton and Company, 1927.

EDITORIALS

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DECEMBER, 1929

THE DIAGNOSTIC VALUE OF MICROSCOPIC EXAMINATION OF FROZEN FRESH TISSUE

THE evolution of the fresh frozen section has been dramatic in its relation to the advance of surgery.

With the old method, preserved material with its artefacts was examined at the end of from 4 days to 2 weeks, at a time when it was of comparatively little use to the patient. Not only does the frozen section make possible an immediate diagnosis, but in malignant disease, through the work of MacCarty and Broders, it gives the grade of malignancy. Knowing the nature of the pathological process, the surgeon has a much better conception of the life expectancy of the patient and is guided in his choice of a radical or palliative operation. For instance, in a malignant growth graded 4 by Broders' classification, the prospects of surgical cure might not balance the immediate risk of the operation, whereas the nature of the growth might be such that other methods of treatment, radiotherapy or heat, would give a far greater chance of recovery.

The history of the frozen section is not concerned with the question of priority of discovery or application of the method, but is merely the story of how it was worked out in one hospital clinic. For the application and success of the method here, credit is due Dr. Louis B. Wilson, who devoted one of his earliest researches to the development of microscopic examination of frozen fresh tissue.

In 1905, Dr. Wilson, who had been an associate in the Department of Pathology at the University of Minnesota, came on to the staff at St. Mary's Hospital in Rochester as chief of the Department of Pathology. Previous to entering on the study of medicine, he had received a long training in physics, biology, and botany. He proceeded to put the pathological laboratories on a scientific basis.

In his work in surgical pathology, Dr. Wilson soon saw the desirability of an immediate microscopic examination of tissues removed at operation and tried out a number of methods of cutting and staining sections for this purpose. His first sections were cut by hand between pieces of elder pith or fixed liver, methods with which he had been familiar as a botanist. His first frozen sections were made by putting the tissues outside the window when the weather was 20 degrees below zero in January, 1905. He then tried out various methods of freezing with ether, with which he had experimented while at the University of Minnesota. In 2 or 3 months, however, he found the method of freezing with carbon dioxide on a Spencer microtome to be the most satisfactory after making some modifications of the freezing chamber of the microtome.

He had been using Bethe's methylene-blue for vitally staining nerve endings for some time and found that it gave excellent results on excised fresh tissue also. Brun's glucose mounting medium he had used years before for botanical preparations.

After getting the method on a satisfactory basis, he used it for several months and then described it in December, 1905, in the *Journal of the American Medical Association*. He published a more complete article in 1915 in the *Journal of Laboratory and Clinical Medicine*. There have been a number of slight modifications of the method since it originally was published, but the essential principles are the same. Perhaps no better results have been obtained by any of the modifications. They have been rather along the line of short-cuts and methods for the preparation of polychrome and methylene-blue.

For years Dr. Wilson checked all his diagnoses made on fresh tissues by the subsequent examination of sections from the same source made from fixed tissues. As one gathers experience this checking is usually unnecessary. Indeed, MacCarty and Broders from their long experience feel as much confidence in their diagnoses on fresh tissue as on fixed.

Pathologists about to begin the study of fresh tissue should always be cautioned on the importance of preparing sections from fresh tissue immediately after its removal, of making several sections of each block so that they may come to know what is a good preparation, and above all, of making a practice of examining blocks from every bit of fresh material available, even though they are certain it is normal, so that they fix in their minds the appearance of normal tissues when stained by this method and thus have a proper background for the recognition of pathological tissues.

Increasing use is being made of this most valuable method of microscopic examination of frozen fresh tissue, which gives a reliable diagnosis from living tissue within 2 or 3 minutes of the time it is removed—in time to be of benefit to the patient. The fresh frozen section has come into its own.

W. J. MAYO

THE PATHOLOGY OF TOXIC GOITER

IT is generally believed (1) that the thyroid shows, in toxic goiter (hyperthyroidism, Graves' disease, thyrotoxicosis, exophthalmic goiter), parenchymatous hypertrophy and hyperplasia; (2) that iodine administered in any form is rapidly taken up by the hyperplastic gland and that involution changes follow, reducing it to a phase analogous to the resting or colloid state; (3) that there is a corresponding amelioration of all systemic manifestations, marked especially by a reduction toward normal in respiratory exchange, heat production, and oxygen consumption (basal metabolism); (4) that these results are but temporary; that in spite of continued iodine administration there is, in most cases, a gradual return of hypertrophy and hyperplasia, with a parallel recurrence of systemic symptoms characterized especially by a rise in metabolism; (5) that the iodine content of the gland is inversely proportional to the degree of hypertrophy and hyperplasia, and that this ratio holds true for all animals from fish to man; (6) that, histologically, nodular goiter is not usually "adenomatous," but that the majority of nodular masses within a goitrous thyroid show little glandular tissue and are in no sense epithelial tumors, or "adenomata," but rather cysts or collections of colloid or circumscribed masses of subinvoluted thyroid.

Over 60 years ago Virchow said, "all so-called varieties of goiter are none other than different terminal metamorphoses of a single type, and a large and striking variety of pathological changes may be combined in the same goiter." Although Virchow, Kocher, and other more recent students of the problem denied the existence of a definite histologic picture in toxic goiter, the view that hyperplasia is constant and specific has been supported by a great many investigators, notably MacCallum, Lewis, Ewing, Farner, and Wilson.

Some newer conceptions of thyroid pathology, although by no means generally accepted, are: (1) Although hypertrophy and hyperplasia are usually associated with toxic goiter, they may be present without associated systemic symptoms. (2) Colloid goiter is a resting stage of hyperplasia, and is preceded by the latter (Marine). (3) Nodular goiter, "involutional bodies," "involutional cysts," etc. (Rienhoff) follow hyperplasia and are invariably preceded by it. (4) Toxic adenoma, "hyperfunctioning adenoma," is an entity distinct from exophthalmic goiter (Plummer) and, *per contra*, toxic adenoma and exophthalmic goiter are but different phases or manifestations of the same disease (Graham, Cutler, Hertzler). (5) Iodine, obviously helpful in the relief of symptoms associated with true exophthalmic goiter, produces no such effect in "toxic adenoma," rather being definitely harmful in such condition. (6) If toxæmia is not already established in an adenomatous goiter, iodine administration will cause an activation of the quiescent gland, resulting in the toxæmia of "hyperfunctioning" adenoma (Plummer) or "iodine hyperthyroidism" (Jackson). (7) Nothing has been discovered to prove that an adenoma, apart from typical hyperplastic Graves' disease, is capable of producing a separate and distinct syndrome in which iodine is less beneficial than in the exophthalmic form (Graham). (8) Exophthalmic goiter and toxic adenoma are clinical variations of a single morbid state (Graham, Warthin). (9) Relatively few so-called adenomata are adenomatous at all, as they form but a small percentage of all goiters (8 per cent, Rienhoff). (10) Deliberate efforts to produce, by the administration of large quantities of iodine, that state referred to as "toxic adenoma" have singularly failed, thus disproving the existence of such an entity as iodine Basedow or iodine hyperthyroidism (Graham and Cutler). (11) All cases of Graves' disease and so-called toxic adenoma possess an identical underlying constitutional abnormality, which is manifested by the constant presence of hyperplastic lymph nodules with large germinal centers. These individuals belong to the "thymicolymphatic diathesis" (Graves' constitution, Warthin), and are, consequently, predestined from birth to hyperthyroidism if the necessary stimuli (physical or emotional stress, infection, etc.) are supplied.

Warthin states that our conception of hyperthyroidism has become an enlarged clinical syndrome, to which innumerable symptoms are daily added until it has become "impossible to distinguish between what some writers regard as exophthalmic goiter and what others call toxic adenoma, and that the pathologist encounters great difficulties if he attempts to apply the histological criteria commonly used for the diagnosis of exophthalmic goiter, hyperthyroidism, or toxic adenoma."

There lurks in all this some truth, much that is difficult of comprehension, some that must be fallacious. We have, however, progressed far, but a solution of the problem of the etiology of goiter probably must be found before thyroid pathology can be more perfectly understood. HAROLD L. FOSS.

MASTER SURGEONS OF AMERICA

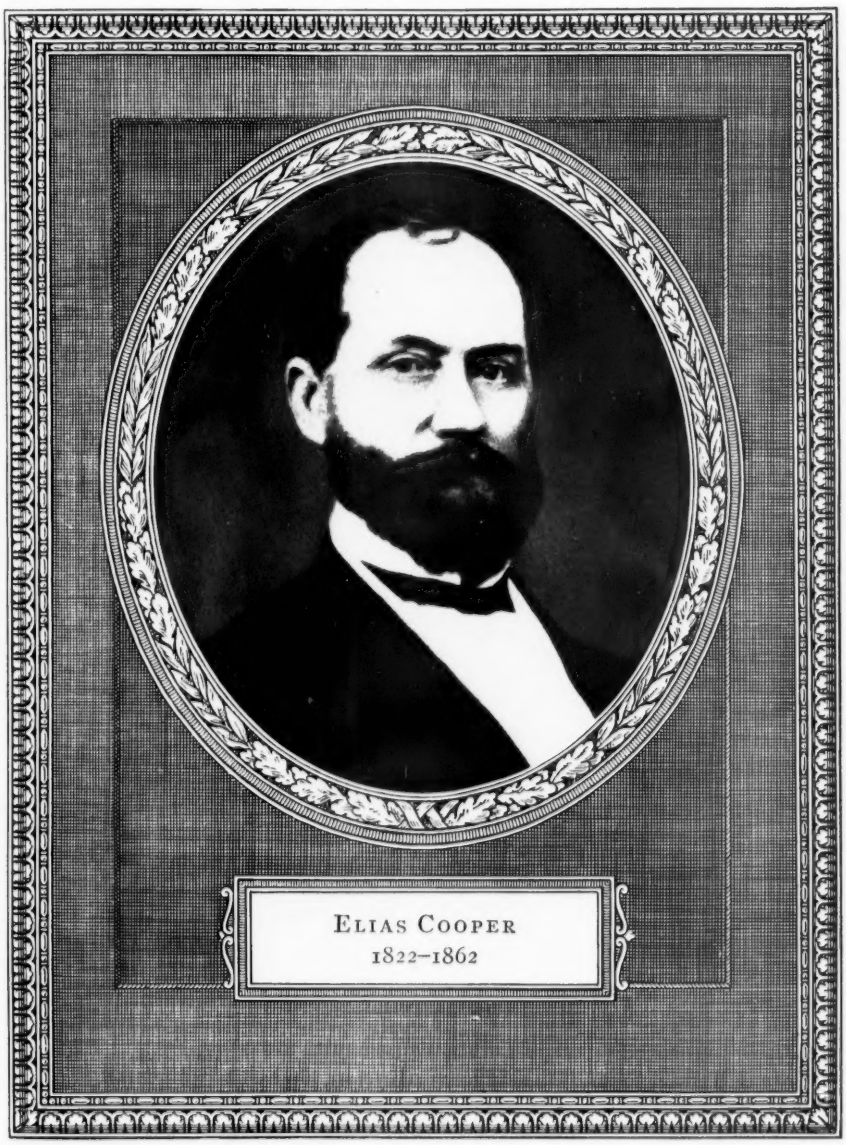
ELIAS SAMUEL COOPER

ELIAS SAMUEL COOPER was emphatically a surgeon. In fact he was known to have somewhat bombastically stated that surgery was the only career worthy of a man. Daring as a surgeon, as a man a fighter, a demon for work, endowed with great originality, he was little short of a surgical genius.

He is generally credited with having been the first, west of Pennsylvania, to have used chloroform as an anæsthetic and was further distinguished by having founded the first medical college on the Pacific Coast. He was a brother of Dr. Esaias Cooper, of Galesburg, Illinois, and of Jacob Cooper, professor of Greek and later of philosophy in Rutger's College, New Jersey.

Born in Somerville, Ohio, in 1822, he began the study of medicine in Cincinnati at the age of 16 years, and was graduated from the St. Louis University 2 years later. He began practice in Danville, Ohio, but in 1844 moved to Peoria, Illinois, where he built the first hospital in that city which he called his "Eye and Ear Infirmary and Orthopedic Hospital," a three story building on what was at that time called the prairie about one mile from the edge of town and was called by the children "The Spook House." His office was in the center of town in one of the very few brick buildings. On the second floor he established an anatomical museum and dissecting room, where he labored far into the night to perfect himself in the anatomy of his operative fields. Doubtless the cadavers were surreptitiously obtained, there being no anatomical law at that date. Some supersensitive citizens resented the row of human skeletons which lined the wall and when the County authorities publicly turned over to Dr. Cooper for dissection, the bodies of two murderers—victims of the first execution in Knox County—a mass meeting of irate citizens was held in protest, but the meeting broke up in an uproar and the matter was quashed. Dr. Cooper, however, found it convenient to arrange for his future dissections at his hospital, which was just out of town.

His life in Illinois was strenuous, to say the least, and the minutes of the Peoria Medical Association have many allusions to Dr. Cooper. On one occasion, in 1850, he was "profoundly reprimanded and requested to resign" because of unethical conduct in advertising his Infirmary. Notwithstanding this criticism, in 1851, he was delegate (alternate) to the American Medical Association. In



ELIAS COOPER
1822–1862

UNIV. OF MICH.

1852, he was secretary of the Illinois State Medical Society, a member of the Committee on Legal Dissection in 1851 and 1852. In 1853 he was president of the Knox County Medical Society.

He presented many papers: e.g., in 1851 he read a paper entitled "Chloroform as an Anæsthetic Agent in 79 Surgical Operations," and in 1852 one on "Treatment of Incomplete Ankylosis of the Knee Joint."

In 1854 he went to Europe visiting various clinics.

In 1855 at the age of 33, Dr. Cooper went to California where he was almost immediately active in the organization of a state medical society. He was said to have been one of the committee who issued the invitation to the medical profession to assemble for that purpose. At the first meeting, 1856, he offered the following preamble and resolution:

> WHEREAS, The laws of our State render surgeons obnoxious to prosecution and liable to heavy damages if they operate wrongfully, through ignorance, at the same time making no adequate legal provision for obtaining a knowledge of the human system; therefore, be it
>
> RESOLVED, That in view of the good of the profession, as well as the community, dissections should be legalized, under all proper restrictions.
>
> RESOLVED, That a committee of five (5) be appointed to memorialize the Legislature upon the subject.

He also read a paper on "Gradual Obliteration of the Abdominal Aorta," having devised an instrument for that purpose and used it with encouraging success in animal experimentation. One animal (dog) lived four days after the occlusion of the aorta was complete. At the second meeting, 1857, Dr. Cooper, being vice-president, presided in lieu of Dr. B. F. Keene (deceased) and gave the presidential address on "Deformities of the Locomotive Apparatus." At the third meeting, in 1858, he read a paper on "Ligating the Satellite Veins in Connection with the Arteries which they Accompany," reporting a case of ligation of the iliac artery with experimental ligation with and without ligation of the accompanying veins in some fifteen dogs.

Dr. Cooper early sprang into prominence in California as a result of an operation for the removal of the breech pin of a fowling piece from beneath the heart. He was the first to perform cæsarean section in California and his patient survived (two cases, in fact, the first leading to a most viciously fought malpractice suit, which, however, was finally won by Dr. Cooper). He declaimed against the accepted doctrine of the danger of the entrance of "atmosphere" into joints, advocated the wide opening and packing with lint of infected joints and those the seat of penetrating wounds. He successfully sutured the fractured olecranon and patella with silver wire, was an enthusiastic advocate of early functional demand in the fractures of the lower extremities, operated for club foot by cutting the contracted soft parts on the short side, as was done by Phelps of New York 40 years afterward, holding the foot subsequently in molded sheet lead, for plaster-

of-Paris bandages had not yet been invented. He announced a new cure for aneurism by cutting down on the sac and sewing it up with needle and thread from the outside. He did much operative work on the eye, even iridectomy and removal of cataract. He successfully removed uterine myomata. He also ligated the innominate artery, his patient living 41 days. He removed a sarcoma of the clavicle taking with it a portion of the sternum.

Much of Dr. Cooper's operative success was due to his free use of alcohol on instruments and hands and parts to be operated on and for the irrigation of wounds, although he was inclined to account for the fact that his wounds did better in California than in Illinois by the difference in climate, or rather that the combination of climate and alcohol had a most remarkably favorable influence in the healing of wounds.

Chloroform was the anæsthetic used—administered on rather coarse sea sponges—perhaps 5 inches in diameter hollowed out on the side toward the patient's face. The mortality was high.[1]

In 1858 Dr. Cooper founded, in San Francisco, the first medical college on the Pacific Coast. In default of a charter an affiliation was made with the University of the Pacific, a Methodist college at Santa Clara, California, by which the school secured the right to issue diplomas. The school received much criticism and ridicule but by persistence, devotion, and good work on the part of the Faculty succeeded in winning the respect of the community.

In the announcement of the first course of lectures to students which began May 12, 1859, occurs the following remarkable proposal for the course in surgery.

> "First, A regular course of lectures on the principles and practice of surgery; second, demonstrative surgery upon the cadaver; third, experimental surgery by vivisection, in which many of the most important principles are indelibly impressed upon the mind of the student. Members of the class are permitted to assist in these experiments upon animals and afterward expected to repeat them under the eye of the professor of surgery. This is an exercise above all others calculated to school the hand, the nerve, and the eye of the pupil, and thereby give him the experience he at once requires in performing the duties of an operative surgeon; a feature in medical education, however, almost entirely neglected in many other medical schools."

Of his own experiments on dogs, the admission of air into the jugular vein and the subsequent resuscitation of the dog by aspiration of the froth from the ventricle is not the least remarkable.

As a result of a controversy with the editor of the *Pacific Medical and Surgical Journal* over the first cæsarean section, and in order to find a medium of publication for his contributions, Dr. Cooper began publishing the *San Francisco Medical Press*, a quarterly journal of medicine and surgery. After Dr. Cooper's death this journal was continued by Dr. L. C. Lane and Dr. Henry Gibbons.

Most of Dr. Cooper's contributions are to be found in the *Transactions of the Illinois State Medical Society*, the *North Western Medical and Surgical Journal*,

[1] Personal communication with exhibition of the sponges by Dr. W. A. Grover who gave many anæsthetics for Dr. Cooper.

California State Medical Journal, 1856, *Transactions of the Medical Society of the State of California*, 1858, *Pacific Medical and Surgical Journal*, and *San Francisco Medical Press*.

Dr. Cooper never married. He died October 13, 1862 at the age of 40, most probably of nephritis. His heart and brain were preserved by Dr. Lane in a kind of shrine in an inner sanctum in Cooper Medical College.

Dr. Cooper was buried in Laurel Hill Cemetery, San Francisco, where a granite shaft carries the simple legend without date

Sacred to the memory of
Elias S. Cooper
Surgeon

The school lapsed in 1864 but was reorganized under the title Medical College of the Pacific in 1870 and later as Cooper Medical College by Dr. Cooper's nephew, Dr. Levi Cooper Lane, who at his personal expense built a commodious building in 1880 renaming the institution in honor of his uncle.

In 1909 the properties of Cooper Medical College were donated to Stanford University and became the nucleus of the newly organized medical department of the University.

EMMET RIXFORD

NOTE.—There is some discrepancy between two records of the date of Dr. Cooper's birth. That given above, 1822, was taken from the obituary notice at the hand of his nephew and associate, Dr. L. C. Lane, in the *San Francisco Medical Press*, 1862, and is presumably the date understood by Dr. Cooper himself. A younger brother, however, gave the date as November 25, 1820.

THE SURGEON'S LIBRARY

OLD MASTERPIECES IN SURGERY

ALFRED BROWN, M.D., F.A.C.S., OMAHA, NEBRASKA

THE NATURAL HISTORY OF PLINY THE ELDER

GAIUS Plinius Secundus, known as Pliny the Elder, was the son of a Roman knight and his wife, the daughter of the senator Gaius Cæcilius. He was born at Comum about 23 A.D., and attained his desire for study after being taken to Rome by his father and educated under P. Pomponius Secundus, the poet and soldier. His education was that of the noble of his day, colored by the decline of Rome, and consequently his book does not show the clear diction of earlier Rome, but evinces a lack of clarity of phraseology which he tries to cover up by ambiguity and extravagant language.

Pliny became the friend of emperors and held many important offices in the Roman Empire. He traveled widely and saw many important events. He studied rhetoric under Seneca, practiced as an advocate, served as a military commander, saw service in many campaigns, and, finally, became præfect of the Roman fleet at Misenum in 77 A. D. Here he was stationed in 79 A.D. when, on the twenty-fourth day of August, Vesuvius erupted and covered the towns of Pompeii and Herculaneum. Though in safety, his desire on the one hand to observe this great cataclysm of nature at closer quarters and on the other to rescue some of his friends, led him to cross the bay and enter the danger zone where he lost his life in his fifty-sixth year.

In spite of his busy life as a citizen and officer of the Roman Empire, Pliny found time for continuous reading and study. His life is an excellent example of the fact that a man with a card catalogue type of brain and endowed with enormous energy can accomplish much. He read incessantly. In the preface of the *Natural History* he claims that he states twenty thousand facts derived from two thousand works that he has read. His nephew, Pliny the Younger, said of his uncle, "He began to work long before daybreak . . . He read nothing extracts." It is said that the notes from which the *Natural History* was written occupied practically one hundred and sixty volumes which must have been indexed and cross-indexed perfectly, for the work was written in two years.

In various parts of the work, Pliny mentions diseases in passing, and shows that he was versed in the common knowledge of the day. He knew quite a bit concerning diseases of the eye; speaks of cæsarean section; mentions an artificial hand of iron to replace one that M. Sergius lost in battle; and describes a case of empyema cured by a stab wound.

In his twenty-sixth book, Pliny discusses new diseases which had lately come to the notice of the people of the Roman Empire, and from his description one would gather that he considered the various forms of leprosy as different diseases and gave them the names of lichen, lascivia, mentagra, carbunculus, and elephantiasis. He says: "The countenance of man perceives new diseases wholly unknown in previous times, not only in Italy but in almost all of Europe. Then they wander much through all Italy, and through Illyric Gaul, or Spain, or elsewhere, even around Rome. One of these diseases is without pain and without danger to life, but of such foulness that death is preferable to whoever has it. This most grave disease is called "lichen," the name in Greek; in the Latin, as it almost always begins on the chin, it was first jokingly called "lascivia" (as is the insolent nature of many in the misfortunes of others); soon it was called "mentagra." The scab in many cases occupies nearly the entire face, the eyes being immune, and descends to the neck, chest, and hand as a foul rash of the skin. This "lues" (pestilence) was not present among our great fathers. It first appeared in Italy in the middle of the reign of the emperor Tiberius Claudius, according to a certain knight, Perusinas, a Roman quaestor, who wrote of it after it had appeared in Asia, whence the contagion was imported to us."

Pliny has been given the credit of being one of the greatest satirists of the medical profession that ever lived. He takes up this phase in the twenty-ninth book: *Concerning the origin of medicine*, etc. In this he gives a short résumé of the history of medicine and discusses the various cults and prominent medical men, especially in relation to their practice with the public and the qualifications of a successful practitioner. He certainly handles the matter without gloves, and one hesitates to say he was wrong, for after reading the various examples that he cites and his different descriptions, one can sum up his general idea in his statement: "People who do not understand Greek place no confidence in a physician unless he practices his art in the Grecian style; indeed, they have less confidence when they understand what serves to cure them."

C. PLINII SECVNDI
HISTORIAE MVNDI LIBRI XXXVII
EX POSTREMA AD VETVSTOS CODICES COLLATIONE
CVM ANNOTATIONIBVS ET INDICE.

BASILEAE
IN OFFICINA FROBENIANA
M. D. XXXIX.

REVIEWS OF NEW BOOKS

THE subject matter of Das's book on obstetrics[1] is treated chronologically and is divided into 11 sections. The first section considers the question whether the ancients possessed any conservative instrument similar to the obstetric forceps of the Chamberlens.

The second section is devoted to the Chamberlens and is a résumé of Aveling's wonderful work. The third section contains, as a natural sequence, the history of the forceps in Holland. The fourth section deals with the eighteenth century forceps.

The subsequent sections deal with successive periods of half or quarter centuries ending with the first 28 years of the twentieth century.

Whenever available, the inventor's own original descriptions have been reproduced. An attempt has also been made to give a short note of their life and work.

Section IX gives a review of the attempts that have been made from time to time to classify forceps. Section X contains chronological lists of forceps, together with a résumé of "a chronology of founders of forceps compiled by Alban Doran." In Section XI, an attempt has been made to collate and record references and illustrations regarding forceps (or founders of forceps) in allegory, literature, and art.

Then follows the appendix of measurements. The list of references form the concluding section.

One may gain an idea of the amount of labor necessary to complete this work when it is stated that there are 2,072 references listed. Everyone interested in obstetrics should read this book.

E. L. CORNELL.

THE author opens the introduction to this small volume[2] with the following statement: "By the term 'Toxemias of Pregnancy' is usually understood a group of disorders associated with gestation, which account for about 26 per cent of the total maternal mortality incident to child-birth. To classify correctly, determine the etiology of, and successfully treat these disorders, has been, and still is, one of the most important problems in obstetrics." He then proceeds to give in detail the changes which occur during normal pregnancy.

The toxæmias of pregnancy are classified under six headings as follows: vomiting of pregnancy, low reserve kidney, nephritis complicating pregnancy, pre-eclampsia, eclampsia, and acute yellow atrophy of the liver.

The etiology of eclampsia is very well written. It is divided into twenty-two subdivisions and the arguments for the belief that the disease is the result of these twenty-two factors is given. We read on and find that the subject is still unclarified. The reviewer had hoped that Stander would clear up the confusion of ideas regarding the etiology of this dreadful disease.

The book is well written and systematically planned. It is a book which everyone doing obstetrics should read and study.

E. L. CORNELL.

"IN offering to the medical world one more treatise on pathology, it would seem necessary, if not to make an apology, at least to give an explanation," say Power and Hala in the preface to their work[3] on this subject. The explanation or apology for presenting this volume of 787 pages to the medical world is that the authors feel that the requirements for a single textbook have not been met from the instructor's standpoint. They have endeavored to present "our knowledge, and particularly our understanding, of pathological processes and changes, concisely and consequentially, concentrating on the diseases that are likely to come within the range of general practice and relegating rarities to brief reference." On the whole, the authors have produced a textbook which fulfills these conditions. It is readable and in general clear. The arrangement is somewhat different from that of the usual textbooks as, for instance, the omission of circulatory disturbances from Part I on General Pathology and the inclusion of them under the Diseases of the Vascular System in Part II, Systematic Pathology. Of the 298 illustrations many are not only diagrammatic but are distinctly crude in their execution, and they certainly do not constitute the most commendable feature of the book. The bibliography is very meager.

While this volume has some merits, they can hardly be said to be so outstanding as to warrant its replacing any one of the several textbooks on the subject already available.

J. P. S.

THIS small volume[4] of 119 pages is a translation of an article by Professor Boveri which appeared some fifteen years ago. The author writes on the problem of the origin of malignant tumors as a zoologist. He admits having had "no experience to speak of in any of the numerous special fields of tumor research." The "essence" of his conception is, "the cell of a malignant tumor is in some way a defective cell; it has lost some qualities of normal tissue cells." The defect inheres in the nucleus. It has not abnormal mitosis but, in general, a definite abnormal chromosome-complex. This thesis is developed largely on the results of study of sea urchin eggs and of other lower forms of life. This

[1] OBSTETRIC FORCEPS; Its History and Evolution. By Kedarnath Das, C. I.E., M.D. Calcutta: The Art Press 1929.

[2] MEDICINE MONOGRAPHS. Vol. XV—The Toxemias of Pregnancy. By H. J. Stander. Baltimore: The Williams & Wilkins Company, 1929.

[3] PRINCIPLES OF PATHOLOGY FOR PRACTITIONERS AND STUDENTS. By H. D'Arcy Power, M.D., F.R.C.S., and William W. Hala, M.D. New York and London: D. Appleton and Company, 1929.

[4] THE ORIGIN OF MALIGNANT TUMORS. By Theodor Boveri. Translated by Marcella Boveri. Baltimore: The Williams and Wilkins Company, 1929.

discussion will be interesting to cytologists and to those who are specializing in cancer research. Its interest to a practicing surgeon will probably be small. J. P. S.

THE third volume of the *Oxford Monographs on Diagnosis and Treatment*[1] by Dr. Henry A. Christian is an authoritative clinical discussion of heart disease. It is, moreover, frank, clear, simple, and practical. It is of interest to note that it does not contain a single electrocardiogram or polygraph tracing. This is characteristic of the bedside point of view emphasized throughout the book. In regard to these he says, "These instruments often help . . . but they are not indispensable, and without them the trained clinician can render to his patients satisfactory service in both diagnosis and treatment."

The intelligent co-operation of the surgeon and internist is frequently mentioned, especially as regards cardiac complexes giving abdominal symptoms, postoperative care, and the surgical treatment of hyperthyroidism. For instance, "It would seem that many surgeons are unaware that cardiac infarction may simulate so closely abdominal lesions . . ." and "Intelligent close co-operation between physician and surgeon is essential for success in the treatment of patients with hyperthyroidism and cardiac disability."

Perhaps the most striking quality of the book is its casting aside of old textbook errors, the reasoned analysis of difficult questions, the sound original advice given on every page; for example, in regard to murmurs, "The American physician still seems so obsessed by the idea of the importance of murmurs that it is difficult for him to admit of cardiac failure without murmurs and equally difficult for him to recognize that an entirely normal heart may show a loud systolic murmur."

I am impressed with the usefulness of this series of monographs which have thus far been received, i.e., the first four volumes out of ten. They are easy reading. They are short, very practical, but complete, in that they are the full personal expression of their authors. This volume on the heart can be read to advantage by everyone—student, practitioner, surgeon, or professor of medicine.

PAUL STARR.

[1] OXFORD MONOGRAPHS ON DIAGNOSIS AND TREATMENT. Edited by Henry A. Christian, M.D., Sc.D., LL.D. Vol. iii—THE DIAGNOSIS AND TREATMENT OF DISEASES OF THE HEART. By Henry A. Christian, M.D., Sc.D., LL.D. New York: Oxford University Press, 1928.

BOOKS RECEIVED

Books received are acknowledged in this department, and such acknowledgment must be regarded as a sufficient return for the courtesy of the sender. Selections will be made for review in the interests of our readers and as space permits.

THE FEMALE SEX HORMONE; PART I—BIOLOGY, PHARMACOLOGY and CHEMISTRY; PART II—CLINICAL INVESTIGATIONS BASED ON THE FEMALE SEX HORMONE BLOOD TEST. By Robert T. Frank, A.M., M.D., F.A.C.S. Springfield, Illinois and Baltimore, Maryland: Charles C. Thomas, 1929.

PRACTICAL LOCAL ANESTHESIA AND ITS SURGICAL TECHNIC. By Robert Emmett Farr, M.D., F.A.C.S. 2d ed. Philadelphia: Lea & Febiger, 1929.

SURGICAL DISEASES OF THE THYROID GLAND. By E. M. Eberts, M.D., with the assistance of R. R. Fitzgerald, M.D., and Philip G. Silver, M.D. Philadelphia. Lea & Febiger, 1929.

AN INTRODUCTION TO THE STUDY OF THE NERVOUS SYSTEM. By E. E. Hewer, D.Sc.(Lond.), and G. M. Sandes, M.B., B.S.(Lond.), M.R.C.S., L.R.C.P. St. Louis: The C. V. Mosby Company, 1929.

THE BLOOD PICTURE AND ITS CLINICAL SIGNIFICANCE (INCLUDING TROPICAL DISEASES): A GUIDEBOOK ON THE MICROSCOPY OF BLOOD. By Professor Dr. Victor Schilling. Translated and edited by R. B. H. Gradwohl, M.D. 7th and 8th rev. ed. St. Louis: The C. V. Mosby Company, 1929.

POSTURE AND HYGIENE OF THE FEET. By Philip Lewin, M.D. The National Health Series. Edited by the National Health Council. New York and London: Funk & Wagnals Company, 1929.

THE MEDICAL RECORD VISITING LIST OR PHYSICIANS' DIARY FOR 1930. Rev. New York: William Wood & Company, 1929

GASTRIC AND DUODENAL ULCER. By Arthur F. Hurst, M.A., M.D.(Oxon.), F.R.C.P., and Matthew J. Stewart, M.B.(Glasg.), F.R.C.P., with the co-operation in the Radiological Sections of P. J. Briggs, M.A.(Cantab.), M.R.C.S., L.R.C.P. New York and London: Oxford University Press, 1929.

ROUTINE PROCEDURES AND FORMULARY; KANSAS CITY GENERAL HOSPITAL. Kansas City: Printing Service Inc., 1929.

THE PATHOLOGY OF THE EYE. By Jonas S. Friedenwald, A.M., M.D., F.A.C.S. New York: The Macmillan Company, 1929.

SYNOPSIS OF MIDWIFERY AND GYNÆCOLOGY. By Aleck W. Bourne, B.A., M.B., B.Ch.(Camb.), F.R.C.S.(Eng.). 4th ed. rev. New York: William Wood & Company, 1929.

EINFUEHRUNG IN DIE GYNÆKOLOGISCHE DIAGNOSTIK. By Professor Dr. Wilhelm Weibel. 4th rev. ed. Berlin: Julius Springer, 1929.

OXFORD MONOGRAPHS ON DIAGNOSIS AND TREATMENT. Edited by Henry A. Christian, M.D., Sc.D., LL.D. Vol. vi—THE DIAGNOSIS AND TREATMENT OF ARTHRITIS. By Russell L. Cecil, M.D., Sc.D. New York: Oxford University Press, 1929.

COMMON INFECTIONS OF THE FEMALE URETHRA AND CERVIX. By Frank Kidd, M.A., M.Ch.(Cantab.), F.R.C.S.(England), and A. Malcolm Simpson, B.A., M.B., D.P.H.(Cantab.); with additional chapters by George T. Western, M.D. and M. S. Mayou, F.R.C.S. New York and London: Oxford University Press, 1929.